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VOLUME 16 NUMBER 6

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OF

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STANDARDS FOR GROWTH AND NUTRITION*

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NEW YORK

The widespread interest lately aroused in the prevalence and degree of malnutrition among school children makes it desirable to examine anew the facts of normal growth in this class of our population. The existence of undernourishment or malnutrition presupposes a normal standard which must be reached if a child is to be considered in perfect health.

The standards by which nutrition has been chiefly measured in the past have been the body-weight and the rate of increase in weight as compared with normal averages. Height and rate of growth in height have also been given a certain amount of value. Most observations have considered both height and weight in relation to the age of the child. The purpose of this paper is to show that the weight-to-age and height-to-age relationships are of comparatively little value in estimating the nutrition of the body. The relation of weight to height is the only one which is reliable.

The standards of nutrition which are susceptible of application to large groups of children are three:

1. The weight-to-height relationship.
2. The annual rate of increase in weight and growth in height.
3. The general appearance of the child.

In the first two of these it is easy to determine the *average;* not so easy to decide what is *normal.* Considerable variations from the average may coexist with normal nutrition. It is important to understand and appreciate how wide the individual variations may be among healthy children of the same race and living in the same environment. What deviation from the average is to be regarded as abnormal, is a question about which differences of opinion may readily exist. Any

* Submitted for publication May 28, 1918.

* Read at the annual meeting of the American Pediatric Society, Lenox, Mass., May, 1918.

standard fixed must be an arbitrary one. To consider as undernourished any child who falls 10 per cent. or more below the normal average is a workable standard and may be adopted in practice until further experience throws more light on the subject.

Since the pioneer work of Quetelet many observations have been made both in this country and abroad on the weight, height and growth of children of the different ages of different races and environment. A special interest attaches to this subject at present, since it is the prevailing opinion that the nutrition of children in all the belligerent countries has greatly suffered because of war conditions and the rise in the cost of food.

In this article will be considered only children of the usual school ages, 5 to 16, inclusive, not because these are more important, but because more data are available than for children of the pre-school age. In the charts given we have, for similar reasons, included only those relating to boys.

During the first year of life we have been accustomed to regard the weight as the chief or sole measure of the progress in nutrition. A regular gain in weight is universally considered as the best evidence of proper growth (an exception of course being made in the case of infants fed on certain proprietary foods and others on mixtures abnormally high in carbohydrates). The growth of the infant in length has been regarded of interest chiefly for the recognition of such conditions as affect the growth of the long bones—rickets, cretinism, chondrodystrophy, etc. After the first year growth of the body in length becomes of more and more interest and of increasing importance in relation to normal nutrition and growth, but it has not received the attention which it should.

Weight and Age Variations.—By far the largest number of observations on weight published have been of weight in relation to age, and the opinion has gained currency that a child of a certain age should have a certain weight to be normal, a moderate latitude for individual variations being allowed. Since most observers give only the *average* weight of children of the different years, it is somewhat difficult from their figures to estimate for the individual variations.

In Chart 1 are given the actual net weights of boys in the Browning School[1] in New York City. It represents 1,774 observations on about 350 different boys whose weights were taken semi-annually, without clothes, over a period of years, the average number of observations on each boy being five. This group of American boys, with but few

1. I wish to express my thanks to Mr. John S. Browning, headmaster of the school, for the use of these data, and also to Dr. C. M. Williams, physical director, by whom most of the weights and measurements were taken.

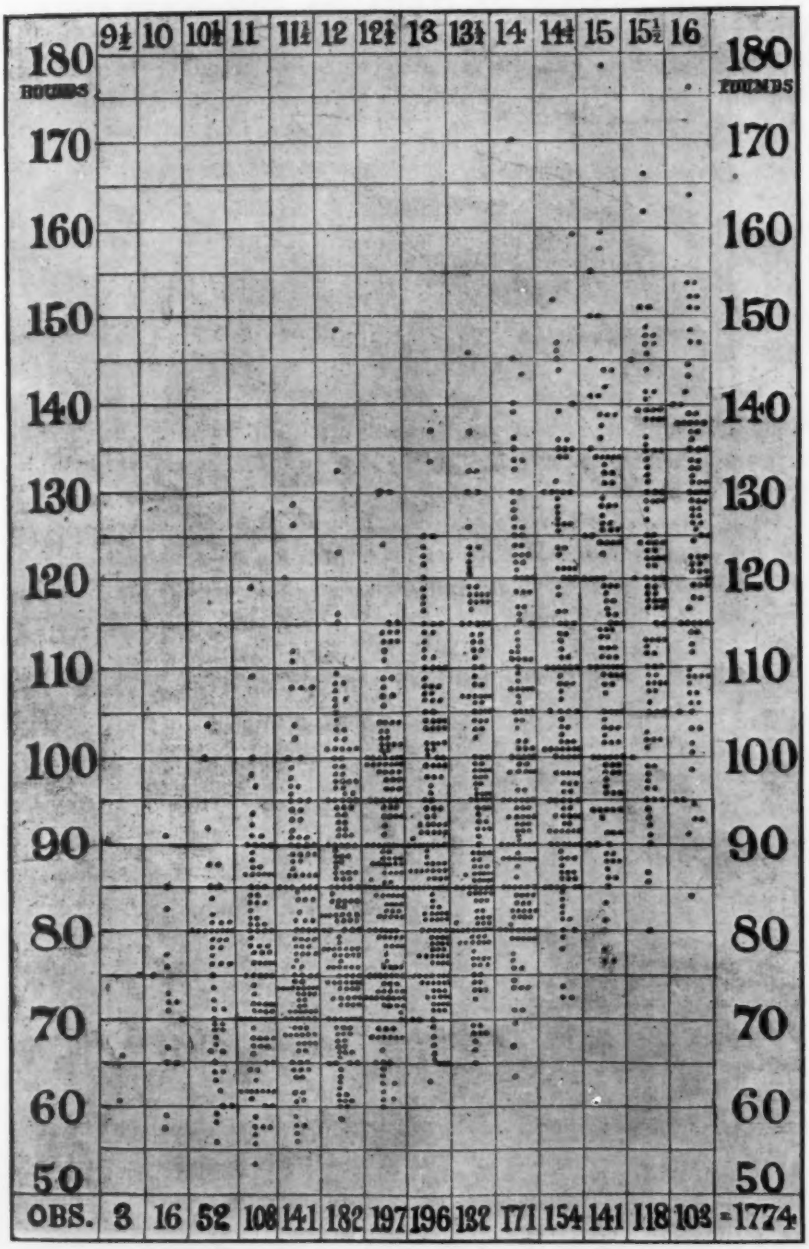

Chart 1.—Age and weight. Boys from the Browning School; 1,774 observations on 350 boys.

exceptions, came from wealthy families and had had the advantage of good care and proper food all their lives. How wide the variations are for the different ages is seen from a glance at the chart. Omitting the few very high and the few extremely low weights, we note that the individual variations for the different years are approximately as shown in Table 1.

TABLE 1.—WEIGHT-AGE INDIVIDUAL VARIATIONS (BROWNING BOYS)

At Age	Range, Pounds	Variation, Pounds	Years' Growth, Approximately
11	60- 90	30	5
12	65-105	40	5
13	70-115	45	5
14	78-125	47	4
15	90-140	50	3½

These extreme variations in healthy boys of the same social class show how hard it is to fix on a standard weight for any age. In this series slightly more than one-fourth the boys were more than 10 per cent. below the average weight of the group for the age.

It is interesting to compare these figures with those of Bowditch[2] for Boston public school boys of corresponding ages. Omitting here also the few extremely high and extremely low weights we have the results set forth in Table 2.

TABLE 2.—WEIGHT-AGE INDIVIDUAL VARIATIONS (BOWDITCH BOYS)

At Age	Range, Pounds	Variation, Pounds	Years' Growth, Approximately
5	32- 52	20	5
6	36- 56	20	4
7	40- 60	20	4
8	44- 64	20	4
9	48- 72	24	4
10	52- 76	24	4
11	56- 84	28	5
12	60- 96	36	4½
13	64-108	44	5
14	68-116	48	4
15	84-128	44	3½
16	96-142	46	3

His observations average about 350 for each year of age. Nearly one-fourth of these boys (23.2 per cent.) were 9.3 per cent. below the average weight for the age, while one-tenth of his boys were 17.2 per cent. below the average weight of the group for the age.

When we come to compare the observations[3] upon weight and age

2. Bowditch: Growth of Children, Public Document, Massachusetts State Board of Health, 1877.

3. The data from which the curves in this paper have been constructed are taken from U. S. Bureau of Education, Bulletin 10, 1914, by Bird T. Baldwin. In this will be found a very extensive bibliography on the growth of school-children.

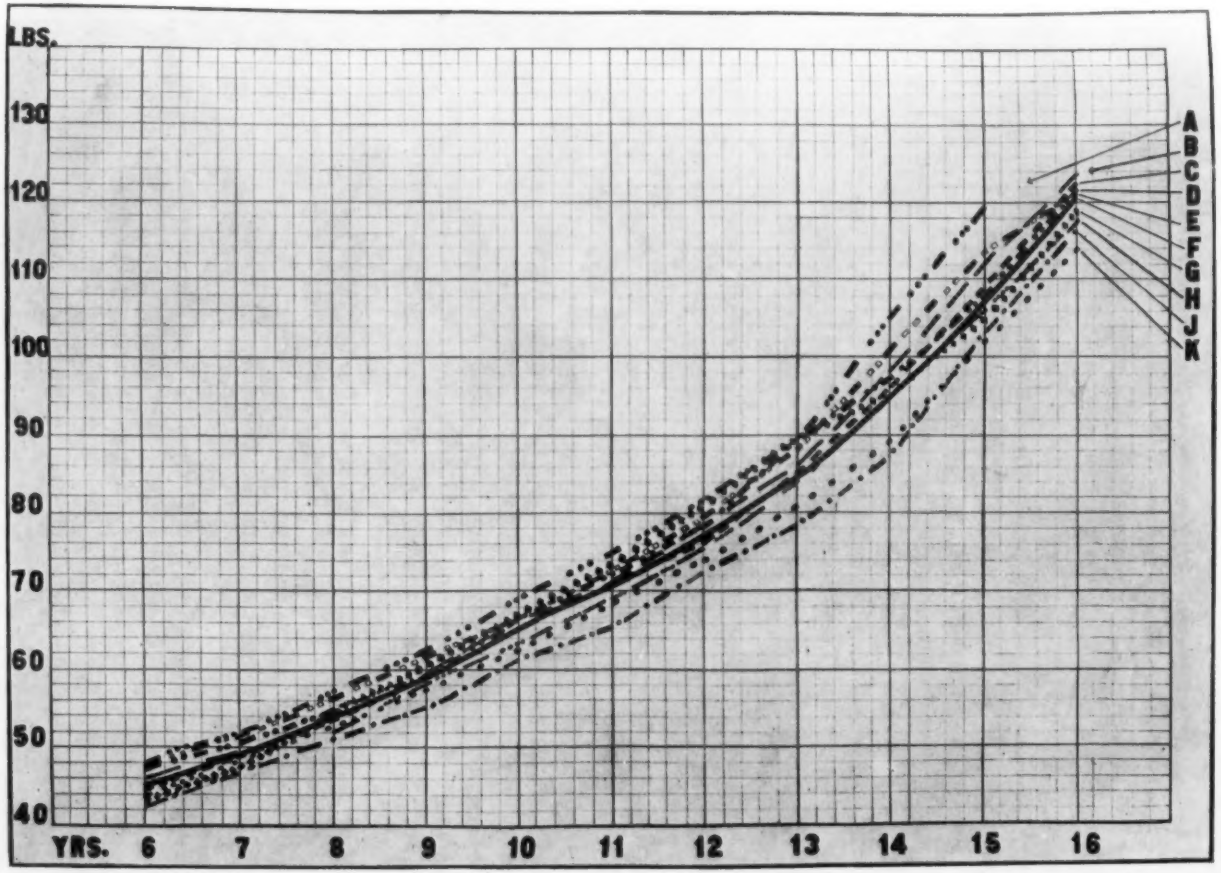

Chart 2.—Weight and age. American boys.

	Approx. No. Observations
A. Boas, California	1,900
B. West, Worcester, Mass.	1,500
C. Peckham, Milwaukee	4,000
D. Barnes, Oakland, Calif.	2,500
E. McDonald, Washington, D. C.	7,000
F. Bowditch, Boston, Mass.	13,000
G. Baldwin, Chicago and New York	100
H. Smedley, Chicago	2,500
J. Hastings, Nebraska	7,000
K. Porter, St. Louis	16,000

made in different parts of the United States and those made in foreign countries with the foregoing we see how wide the average weight-age variations are.

In Chart 2 are given the average weight curves of ten different series of observations on American schoolboys made in widely scattered parts of the country. These represent in all nearly 50,000 observations. The lowest average curves are "J," that of Hastings, and "K," of Porter; the highest average is "A," that of Boas. The solid black line, "F," of Bowditch, is very nearly the mean of the entire group, and may therefore perhaps be taken, as well as any single curve can, as the average curve of the country as a whole.

It will be noted that the variation in these curves, which represent averages, at 6, 7 and 8 years is only about 5 pounds; also that this variation steadily increases with the age, so that at 14 and 15 years it is about 15 pounds. There is, however, a general parallelism to the curves, indicating that the rate of gain is nearly the same in the different groups.

When Chart 2 is compared with Chart 3 it is evident what a large part nationality plays in modifying a weight-age curve. The observations in Chart 3, like the others, were made on boys in public schools. The heaviest boys for their ages are Russians and Polish (A and B). The lightest are Japanese (H); then in order come the Italian in Italy (G); Italian in the United States (K), and the German (J). Even omitting the Japanese, we see how wide is the variation in the weight-age curve among the European nations, all of which are represented in our present American population. With such wide variations as those noted both in foreign and in American boys it is evident that weight for age is not of great importance in determining the nutrition of a child.

Height and Age Variations.—The curves for height and age in a general way correspond to those of weight and age among the different groups in the United States and also among the foreign boys in their relation to each other, but the variations in the height-age curves are considerably narrower.

Chart 4 shows the variations in a group of boys of the same nationality and the same social environment — the boys of the Browning School. Omitting the few extremely high and extremely low measurements we note in Tables 3 and 4 the range and variation in height.

Both these groups show how wide are the individual variations met with in boys of the same general class. By comparing these tables with the weight-age Tables 1 and 2, it will be seen that, estimating in years' growth, the differences in weight among individuals are less

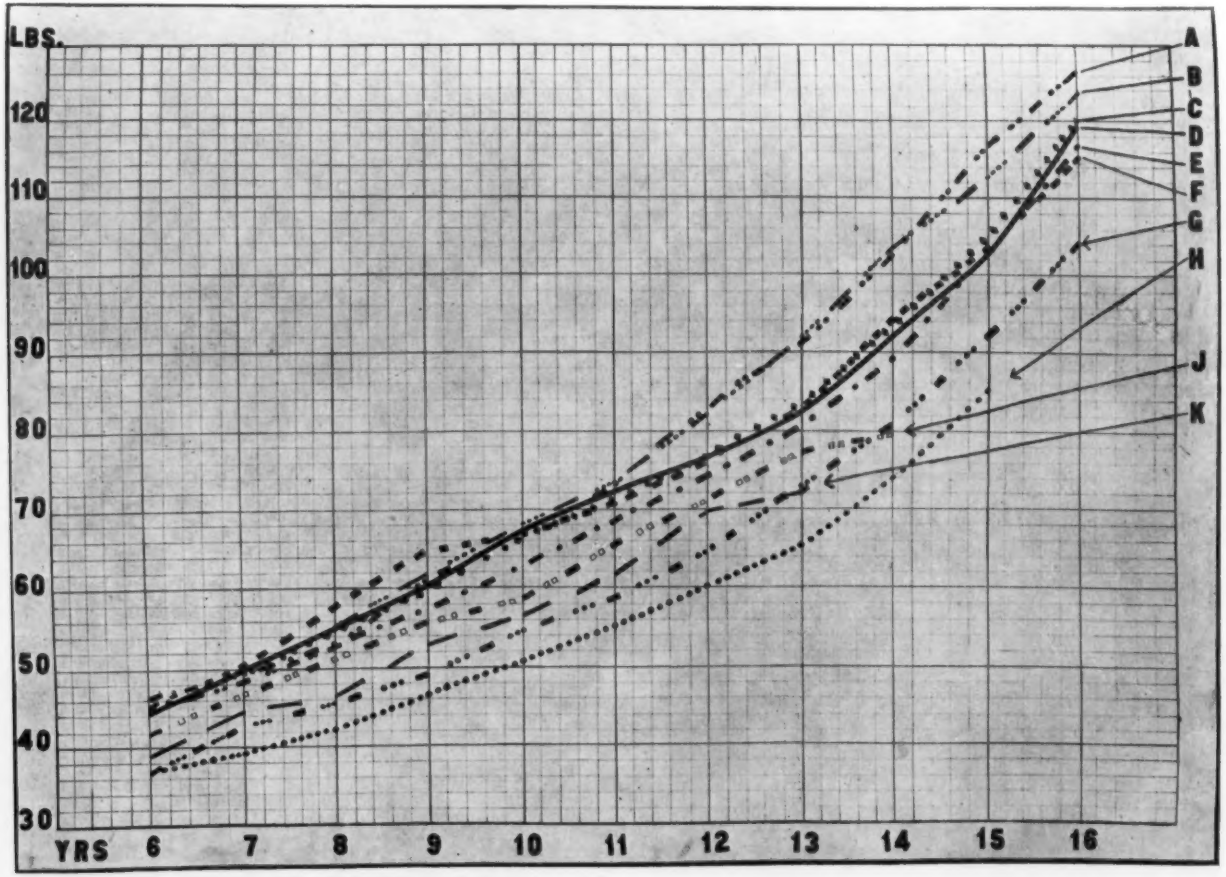

Chart 3.—Weight and age. Foreign boys.

	Approx. No. Observations
A. Erismann, Russian	2,450
B. Suligowski, Polish	1,250
C. Stevenson, British	.....
D. Roberts, British	25,000
E. Hertel, Danish	17,000
F. Key, Swedish	14,000
G. Pagliani, Italian in Italy	900
H. Misawa, Japanese	9,000
J. Schmidt, German	4,700
K. Hrdlicka, Italian in U. S.	.....

than the differences in height, and that in both weight and height as boys approach maturity these differences tend to become less.

In Chart 5 are given the height-age curves of the American boys whose weights are given in Chart 2. It will be seen how closely these curves of average height of different groups correspond.

Only one, "A," Baldwin's, differs in any important degree from the average; "K," Porter's curve, is a little lower than the others; and "B," West's curve, a little above. The curves of the other seven observers are almost identical. The solid black line, "D," is the curve of Bowditch. This line may therefore be taken as a fair average of school boys of the United States.

TABLE 3.—HEIGHT-AGE INDIVIDUAL VARIATIONS (BROWNING BOYS)

At Age	Range, Inches	Variation, Inches	Years' Growth, Approximately
11	53-60	7	3
12	55-62	7	3
13	58-64	8	2½
14	59-67	9	2½
15	61-70	9	2
16	62-72	8	2

TABLE 4.—HEIGHT-AGE INDIVIDUAL VARIATIONS (BOWDITCH BOYS)

At Age	Range, Inches	Variation, Inches	Years' Growth, Approximately
5	38-44	6	3
6	40-46	6	3
7	42-49	7	2½
8	44-51	7	2½
9	46-53	7	2½
10	48-55	7	2½
11	49-57	8	2½
12	50-60	10	3
13	52-62	10	3
14	54-65	11	3
15	57-67	10	2½
16	60-70	10	2½

In Chart 6 are given the curves showing average heights at the different ages of various foreign groups of boys. The variations are almost as great as are seen in the average weights of these boys, and in a general way the curves of weight and height correspond. It is clear, then, that in height as well as in weight, race must be taken into account in determining the normal average.

When very large groups are considered, the increase in height and in weight goes on together proportionately in children who are perfectly healthy. When smaller groups or individuals are closely observed it is seen that growth in height is, with the majority, more irregular than the increase in weight and is influenced by many conditions about which as yet we know very little. In private practice

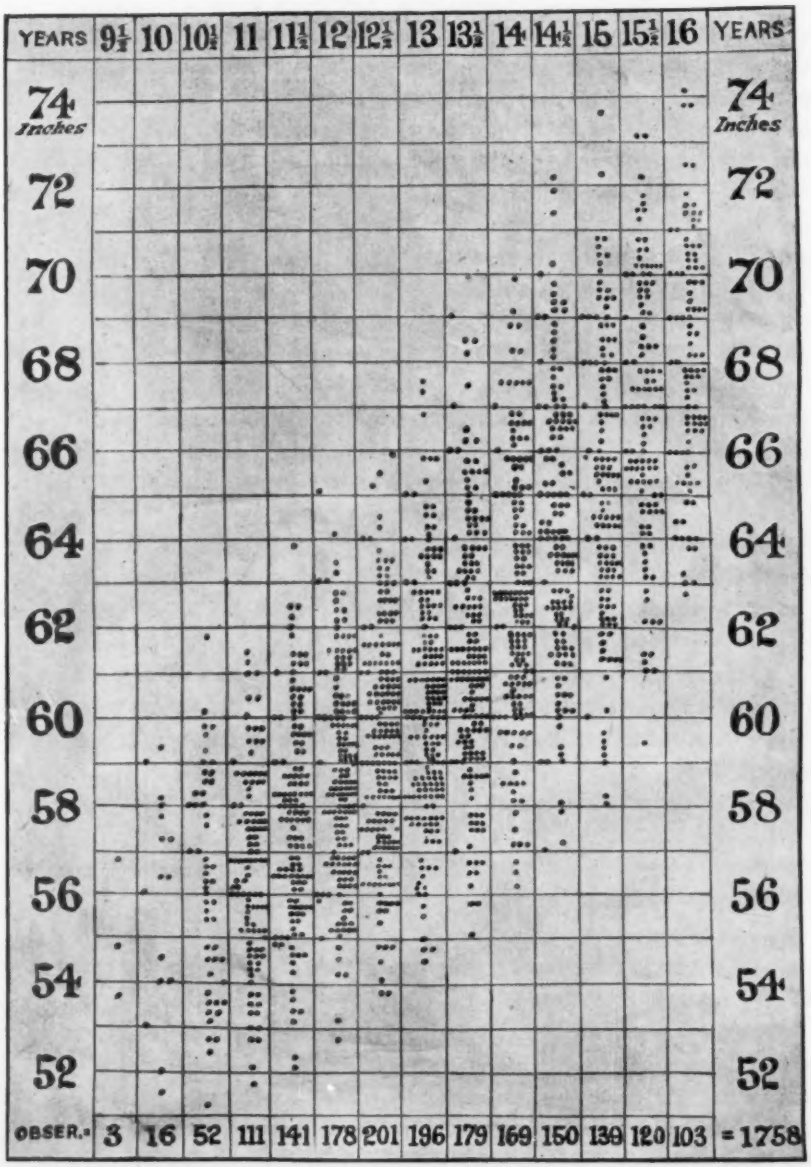

Chart 4.—Age and height. Boys from the Browning School; 1,758 observations on 350 boys.

one is constantly impressed with the great influence of heredity as affecting growth in height. This is a factor the importance of which it is difficult to estimate when large numbers of school children are measured. The variations in height are so great that it is difficult to say how wide a deviation from the average may be considered abnormal even in the same race and social status. In the young the impulse to grow is so strong that even when food is insufficient it may continue, the increase in height taking place at the expense of the stored-up materials in the body. It may be coincident with no increase in weight or even at times with actual loss in weight. As a guide to a child's nutrition, height-age curves are therefore quite unreliable.

Weight-to-Height Variations.—A much more important relationship than weight-to-age or height-to-age as an indication of the state of the child's nutrition is that of the weight-to-height. The value of this is recognized by all the insurance companies. What an adult man or woman should weigh for his or her height is brought to our notice constantly by the figures given on the penny-in-the slot scales. In childhood less attention has been paid to this relationship than its importance deserves, and even though here there are no absolute figures of weight-to-height which can be regarded as normal, and there are considerable individual variations seen in healthy subjects, yet these variations from the average are not so great but that they furnish important information regarding the nutrition of the child.

Considering how wide is the variation seen in different races in the height and weight of boys of the same age, it is rather surprising to see how nearly alike are the curves of weight-to-height. This is shown in Chart 7, which gives the weight-to-height curves for boys from five different nationalities; the two tall and heaviest (Russian and Polish), the two small and lightest (Italian and Japanese) and the American, using Bowditch's figures for Boston school boys of American parentage.

That these curves are so nearly alike indicates how independent of nationality is this index of nutrition. Thus, the average Japanese boy of 50, 60 and 80 pounds is only one inch shorter than the average American boy of the same weight; while those of 70 pounds weight appear exactly the same height. So, also, the Italian boy, who, though much shorter than the American for his age (Chart 5), is for his weight only from half an inch to an inch shorter than the American up to a weight of 105 pounds and a height of 62 inches; the only exception to this being the short period when the weight is in the neighborhood of 50 pounds and the height near 50 inches. The chief difference between the tall and short races is that in the latter growth is slower and ceases at an earlier period.

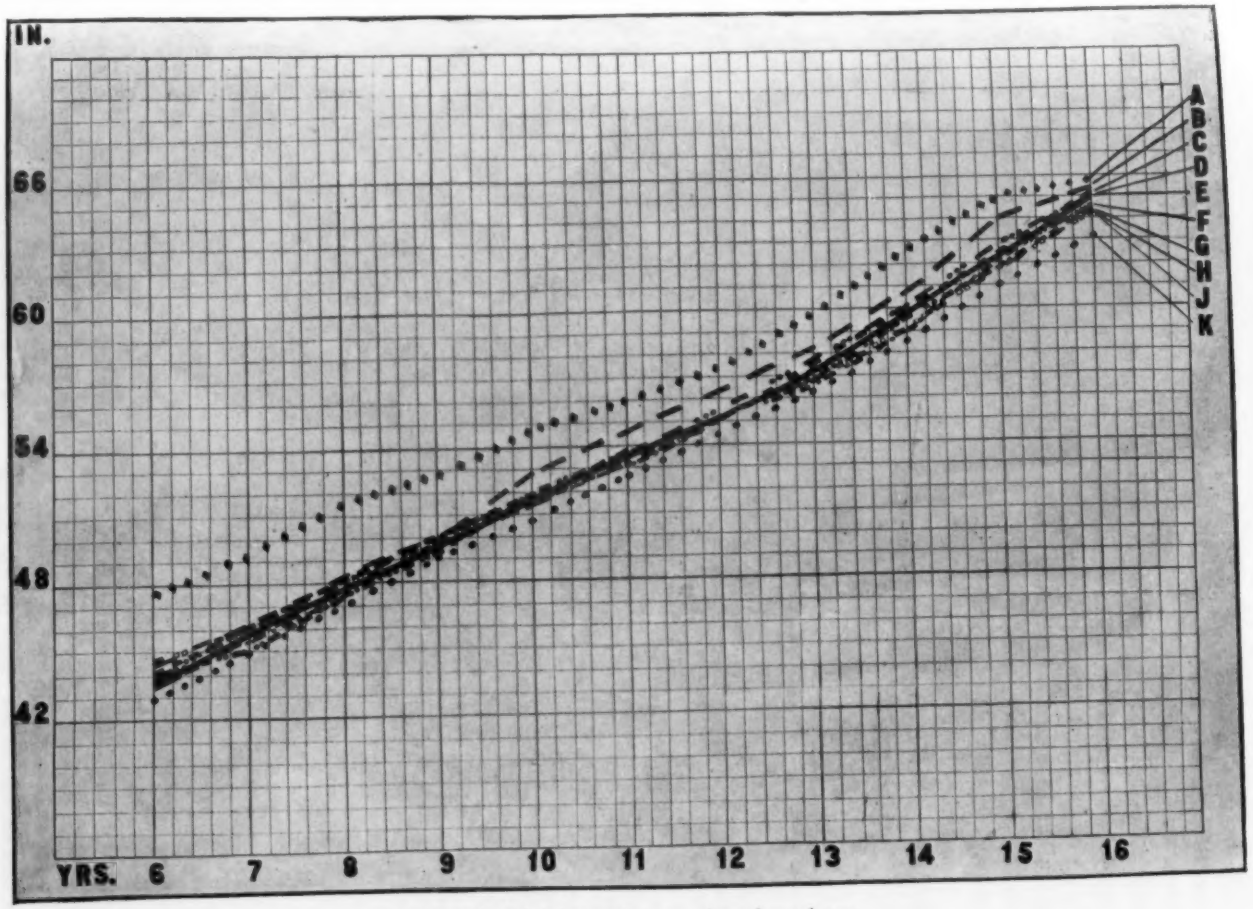

Chart 5.—Height and age. American boys.

	Approx. No. Observations
A. Baldwin, Chicago	300
B. West, Worcester, Mass.	1,500
C. Peckham, Milwaukee	4,000
D. Bowditch, Boston	13,000
E. Boas, California	44,000
F. Smedley, Chicago	2,500
G. Barnes, Oakland, Calif.	2,500
H. Hastings, Nebraska	7,000
J. McDonald, Washington, D. C.	7,000
K. Porter, St. Louis	15,000

Weight-Height Index.—The determination of where a child stands in the relation of his height to his weight is undoubtedly the best simple guide in fixing his status as to his nutrition. This ratio expresses the general solidity or robustness of the body and is therefore significant. The weight in kilos divided by the height in centimeters, or weight in pounds divided by height in inches gives what is sometimes called the *weight-height coefficient* or *weight-height index* of the individual. This weight-height index increases steadily and quite uniformly from 5½ up to 18 years. According to the observations of Baldwin, the lowest weight-height index in boys is at 6 years, when it is 0.15; the highest is at 17½, when it is 0.40.

The same thing is shown in another way in Table 5, the data for which are taken from Bowditch. Here is given the increase in weight which corresponds to each inch increase in height.

TABLE 5.—WEIGHT-TO-HEIGHT INCREASE

	Boys			Girls		
Height, Inches	Weight, Pounds	Increase Per Inch, Pounds	Approx. Age, Years	Weight, Pounds	Increase Per Inch, Pounds	Approx. Age, Years
42	41.8	...	5	40.9	...	5
43	43.6	1.8	..	42.6	1.7	..
44	45.6	2.0	..	44.5	1.9	..
45	47.6	2.0	6	46.5	2.0	6
46	49.6	2.0	..	48.5	2.0	..
47	52.1	2.5	7	50.7	2.2	7
48	54.6	2.5	..	53.2	2.5	..
49	57.3	2.7	9	56.1	2.9	9
50	60.2	2.9	..	58.8	2.7	..
51	63.2	3.0	10	61.4	2.6	10
52	66.3	3.1	..	64.4	3.0	..
53	69.2	2.9	11	67.5	3.1	11
54	72.7	3.5	..	71.0	3.5	..
55	76.4	3.7	..	74.9	3.9	..
56	80.2	3.8	12	78.8	3.9	12
57	84.0	3.8	..	83.4	4.6	..
58	87.9	3.9	13	87.9	4.5	..
59	91.6	3.7	..	93.3	5.4	13
60	95.5	3.9	..	98.8	5.5	..
61	100.5	5.0	14	105.4	6.6	..
62	105.6	5.1	..	115.7	10.3	14
63	110.7	5.1	15	.....	....	..
64	115.9	5.2	..	....	....	..
65	121.0	5.1	16	.....	....	..
66	126.6	5.6	..	.....	....	..
67	136.1	9.5	..	.. ..	....	..

In all observations on weight and height of children the *average* is not to be confused with or regarded as the ***normal***. There are considerable variations on either side of the average which should be regarded as within the range of the normal. The normal is a zone, not a line. How far below the average weight for his height a child should be for his nutrition to be considered below the normal, one cannot say

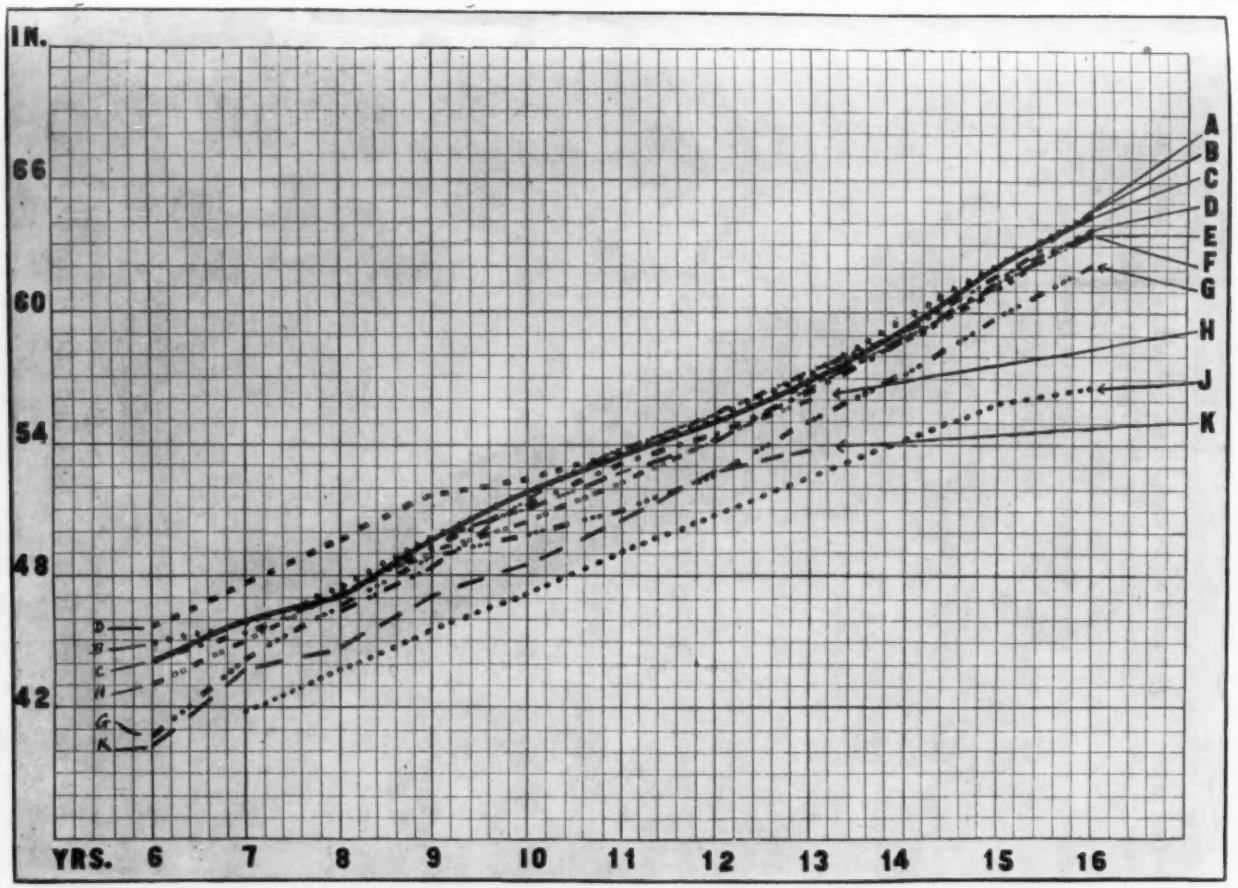

Chart 6.—Height and age. Foreign boys.

	Approx. No. Observations
A. Hertel, Danish	17,000
B. Stevenson, British	.....
C. Roberts, British	25,000
D. Key, Swedish	14,000
E. Erismann, Russian	3,000
F. Suligowski, Polish	1,000
G. Pagliani, Italian in Italy	900
H. Schmidt, German	4,700
J. Misawa, Japanese	9,000
K. Hrdlicka, Italian in U. S.	.....

absolutely. If we regard all those who are 10 per cent. or more below the average line as undernourished or as examples of malnutrition, we shall not go far wrong. For practical purposes we may assume this to be correct.

Annual Rate of Increase in Weight and Height.—The weight to height relation we have concluded to be the best general starting point or base line for observations on the nutrition of children;[4] but nothing indicates their progress so well as the annual increase in height and weight. Like an infant, a boy or girl to be healthy must grow; and a record of such growth of any child extending over a period of years will be found to be an accurate and trustworthy record of his general health during that period.

The annual rate of gain of healthy school boys of corresponding ages is nearly the same in different parts of the United States and in foreign countries. In the accompanying table (6) is given the average annual gain in weight and height of American boys and girls, calculated from the data represented in the curves previously given. It will be noted that in the boys the gain in weight increases with each year of age after 7 years, except between 10 and 11, when it is less than for the preceding period. This slowing up in growth before the marked increase of approaching puberty is noted in nearly all the different groups of boys observed.

The yearly increment in height is less between the tenth and twelfth years than before or after this period. Like the increase in weight, it is least between 10 and 11 years. It does not show a marked increase until after 12, when the rapid growth of puberty begins. This is about a year later than the more rapid increase in weight.

As children grow older the increase in height and weight is less uniform and regular. There are seen with nearly all healthy children periods, often of several months' duration, in which growth and gain in weight are rapid, followed by periods in which both are much slower. This may occur where no special cause can be discovered. It is often seen as puberty approaches.

Children who are much below normal weight usually gain rapidly when the cause for the low weight can be discovered and removed.

Seasonal Growth.—The question is often discussed during what season of the year the most rapid growth in height and increase in weight occur. The observations made on the Browning boys are of interest as throwing some light on this point. Of 703 observations

4. There are some exceptions to this rule. Certain children, not a large group, may be stunted in growth by conditions which also affect weight, so that the relation of weight to height is average, though the child is much below the normal in both.

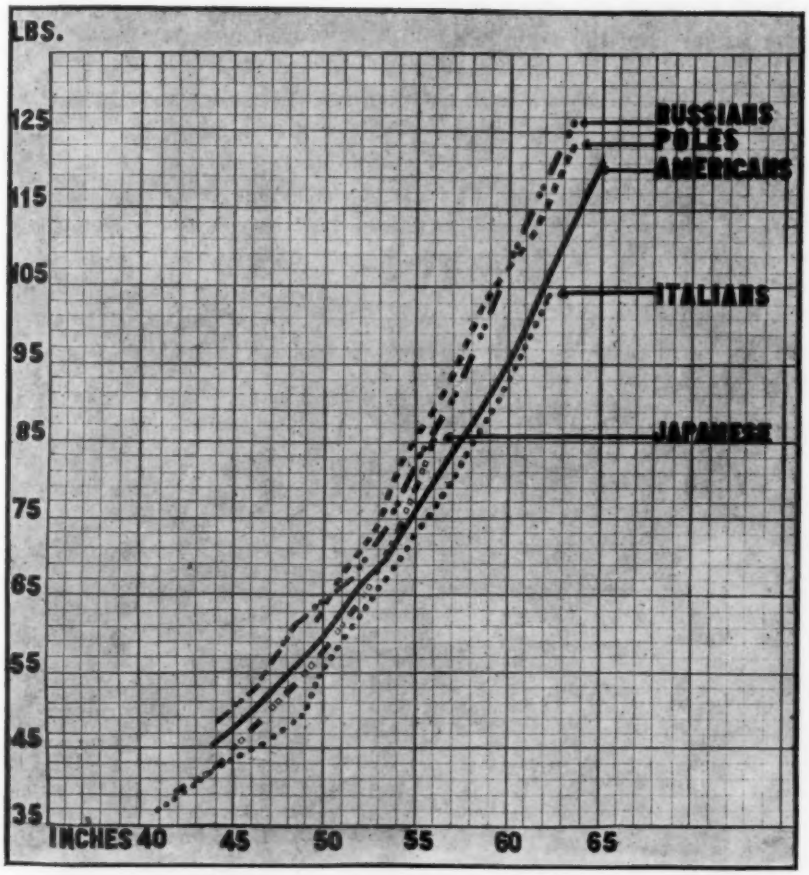

Chart 7.—Weight and height.

made on weight for the six months from November to May and from May to November, the average gain for the summer six months was 1¼ pounds greater than for the same boys during the winter six months. The same thing was seen in growth in height. Of 544 observations made on height, the average increase was 0.36 inch greater for the summer six months than for the same boys during the winter six months. These facts confirm the general law that in healthy individuals increase in weight and growth in height go on together and approximately at the same rate.

TABLE 6.—AVERAGE ANNUAL INCREASE IN WEIGHT AND HEIGHT AT THE DIFFERENT AGES

BOYS

	Weight		Height	
Age, Years	Range, Pounds	Average, Pounds	Range, Inches	Average, Inches
6- 7	3 - 5	4	1.5-2.5	2
7- 8	3.5- 5	4.75	1.5-2.5	2
8- 9	4 - 6	5.25	1.5-2.5	2
9-10	4 - 7	6	1.5-2.5	2
10-11	4 - 6.5	5	1.4-2.0	1.7
11-12	4.5- 7.5	6.5	1.3-2.0	1.8
12-13	6 - 9	8	1.5-2.5	2
13-14	8 -12	10	2 -3	2.5
14-15	10 -15	12.5	2 -3.5	2.7
15-16	9 -15	13.75	2 -3.5	2.7
16-17	4 -12	6.5	0.5-2	1.2
17-18	4 - 8	5	0 -1	0.5

GIRLS

	Weight		Height	
Age, Years	Range, Pounds	Average, Pounds	Range, Inches	Average, Inches
6- 7	3 - 5	4	1.5-2.5	2
7- 8	2 - 5	4.5	1.5-2.5	2
8- 9	3.5- 6	5	1.3-2.4	1.75
9-10	4 - 6	5.25	1.5-2.5	2.25
10-11	4.5- 7	6.5	1.5-2.5	2
11-12	7 -11	9.5	2 -3	2.5
12-13	8 -12	10.5	2 -3	2
13-14	7 -11	9.5	1.3-2.4	2
14-15	5 -10	7.5	1 -2	1.25
15-16	3 - 8	6	0 -1	0.75
16-17	2 - 6	3.5	0 -0.5	0.50
17-18	0 - 2	0.5	0 -0.2	0.2

The explanation for more rapid growth during the summer season would seem to be that this period includes the school boy's vacation time, during which he leads a more free and more outdoor life; while the winter period includes most of the colds and other respiratory diseases and a large part of the contagious diseases from which those of these ages are likely to suffer, all of which arrest growth and gain in weight.

General Appearance.—In estimating a child's condition as to his nutrition, careful inspection may tell a good deal, particularly to an experienced observer. The color of lips and cheeks and the facial expression are significant of anemia, fatigue or even exhaustion. If the clothing is removed to the waist, the muscular development and the amount of subcutaneous fat can be determined as well as a narrow chest and round shoulders recognized. Such an inspection taken in connection with height and weight enables the examiner to form a pretty accurate estimate of the child's nutrition.

The so-called Dunfermline scale in which children are graded in their nutrition by their appearance as I, good; II, fair; III, below average; IV, very bad, is not a very trustworthy way of estimating the nutrition if such inspection is made without undressing the child at all and without taking account of height and weight. But taken in conjunction with height and weight and when made by a person of experience, the general appearance is of great assistance in grading the child, and the grouping in the above scale is very convenient for practical purposes.

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EXPERIMENTAL RESEARCH ON THE EFFECT ON THE GASTRO-INTESTINAL TRACT OF THE EXCRETION OF POISONS, CONSIDERING THE AGE OF THE ANIMALS*

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It is well recognized that a part of the poisons absorbed by the blood is excreted in the gastro-intestinal tract and produces a certain effect on its mucous membrane. However, systematic histologic experiments concerning these effects are lacking, and especially as regards the differences of effect between grown and very young animals.

From my experiments I expected: (1) that the various kinds of poisons offered would produce different effects on the different regions of the intestinal tract; (2) that the pathologic-anatomic and pathologic-histologic study of these effects would show various results, depending on the kind of poison used; (3) that in such experiments the results obtained would differ to a certain degree in very young animals from those in grown animals.

It is certain that in diseases of the intestinal tract of infants the clinical signs and the pathologic-anatomic findings usually differ from those of the adult. For instance, we know that nutritional disturbances or the enterides of infancy are not localized in the intestine, as is the case with adults, but are accompanied by a general affection, causing severe disturbances in the general metabolism; however, contrary to expectation, the postmortem examination shows no such violent changes in the intestinal mucosa. Again, we know that dysentery, an infectious disease of the intestinal tract, often runs in infancy an atypical course, and pathologic-anatomic changes are not distinctly localized in the large intestine, as is usually the case in the adult, but often extend to the small intestine.

Experimental pathologic research concerning the above points seems to be lacking. In my experiment it was purposed to compare the function of the gastro-intestinal mucosa, in regard to the excretion of poisons, and the change produced thereby, in the intestinal mucosa of very young animals with that found in grown animals.

METHOD

The poisons used were, 1 per cent. colloidal bismuth in watery solution; mercury (chiefly from 0.5 to 1 per cent. sublimate sodium chlorid solution); 1 per cent. colloidal silver solution; from 0.1 to 1 per cent. arsenous acid solu-

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tion; from 0.3 to 5 per cent. potassium bichromate solution; ricin in physiologic salt solution (0.5 mg.: 10); from 0.2 to 0.5 per cent. cantharidin in olive oil; from 0.5 to 1 per cent. snake poison in glycerin-water solution; sterile emulsion of dysentery (Shiga) bacilli; paratyphoid B; typhoid, and cholera bacilli. These bacilli were cultured in agar mediums for twenty-four hours, and were emulsified in a physiologic salt solution in the proportion of 5 c.c. to one agar tube, and were sterilized for one hour at 60 C.

These poisonous solutions were injected intravenously or subcutaneously into rabbits (grown and very young), white rats (grown), dogs (grown, and very young), chickens (grown, and very young), and pigeons (grown).

The feeding of the animals was arranged according to their physiologic habits. The very young rabbits were nursed twice a day by the mother animals. The very young dogs were fed with cow's milk.

As regards the doses of poisons used, the animals were usually poisoned with a dose in proportion to their weight; however, because the disposition of each animal varied according to kind and age, a dose was often given from which the animal became distinctly prostrated, within a certain number of hours or days.

Method of Examination.—The animals were usually killed and dissected at various stages, such as in the acute stage of poisoning (from a few hours to a few days), or in the subacute or chronic stage (from a few days to usually one or two weeks).

Microscopic sections were made from several parts of the stomach, small intestine, cecum and large intestine, and were generally stained with hematoxylin and eosin. Sometimes a frozen section was examined by itself, or stained for fat by sudan III and sometimes by iron reaction. Moreover, I contrasted the intestinal tracts of normal animals of various ages.

EXPERIMENTS

1. *Bismuth Experiment.*—Findings in *grown animals* (6 rabbits, 4 white rats, 3 dogs, 5 chickens, and 3 pigeons): The important change in the intestinal tract was a brownish-black pigmentation in the mucous membrane of the cecum and large intestine, especially intense in the cecum of the rabbit, rat, and chicken, and in the large intestine and cecum of the dog. Moreover, in the cecum of the rat and in the large intestine and cecum of the dog, a necrotic inflammation was produced in part of the mucosa.

Microscopic examination of frozen sections of these pigmented areas showed a pigmentation in the tissue of the mucosa. The epithelial cells on the ends of the villi contained fine, more or less irregular, brownish-black granules in the protoplasm on the superficial side of the nucleus. The capillary walls near the apex of the villus were brownish-black in color; large migratory cells filled with coarse brownish-black pigments were found here and there in the membrana propria and between the epithelial cells. In some cases, in which the animals were poisoned by large doses, similar pigmentation, but very slight, was also seen in the mucosa of the stomach, especially in the fundus, and just a trace was, rarely, found in the small intestine.

Sawai[1] has proved chemically that this pigment is a sulphur compound of bismuth, and he has shown experimentally that it is the result of excretion, not of resorption.

From the sections stained with hematoxylin-eosin a degeneration and necrosis of the epithelial cells occurred showing granulated or vacuolated protoplasm and figures of karyorrhexis or karyolysis, together with a slight infiltration of round cells in the distinctly pigmented regions. In cases of dogs and rats, a necrosis of the tissue of the mucosa was seen, with some hemorrhage and moderately intensive infiltration in the submucosa, in the lower portions of

1. Sawai: Kyoto Igakuzassi (Japanese), 1916, No. 5.

the intestinal tract. Some catarrhal changes were occasionally found in the mucosa of the stomach and small intestine.

Findings in *very young animals* (18 rabbits ranging from 3 to 18 days of age, a dog 3 days old, 7 chickens ranging from 3 to 10 days old): In most cases a slight brownish-black coloring was produced in the mucous membrane of the stomach and small intestine, especially distinct in the fundus and jejunum, sometimes extending as far as the large intestine, but here much slighter.

Microscopic examination showed that the pigmentation of the mucosa was more intensive in the stomach and small intestine than in the lower portions.

The mode of excretion of the bismuth was so characteristic that the excreting granules were found moderately diffuse, not only in the surface epithelium, but often in the glandular epithelium of the mucosa; while in cases of grown animals the metal was excreted principally and in more concentrated form by the epithelium on the ends of the villi in the lower portions of the intestine. The pigmentation of the mucous membrane in the cecum and large intestine of very young animals, however, was more intensive in comparison with the findings in the stomach and small intestine of grown animals.

The capillary walls were colored, the pigmentation of the migratory cells was rarer and slighter than in grown animals.

In animals about 3 weeks of age the pigmentation in the lower portions appeared more intensive and was slighter in the stomach and small intestine. In animals 2 months old the findings were similar to those of grown animals.

These different findings, comparing very young animals with grown animals, may be due to the scarcity of sulphur in the intestinal contents of the former. To prove this the following experiment was performed:

An ammonium sulphid solution, or flowers of sulphur, was given to several groups of very young and grown rabbits by esophageal catheter twice a day, and in some cases, also, at the same time *per ani*, soon after the injection of bismuth. On the third or fourth day after this treatment the animals were killed and examined. The pigmentation of the mucosa was found in the very young animals to be most intensive in the stomach and small intestine; while in grown animals the lower region was more distinctly pigmented.

Hence, these differences must have been chiefly due to the different modes of intestinal excretion, according to the age of the animals.

2. *Mercury Experiment.*—Findings in *grown animals* (9 rabbits, 3 white rats, 3 dogs, 6 chickens, and 3 pigeons): The important changes were catarrhal inflammation in the first stage, hemorrhagic inflammation in the second stage, and necrotic inflammation in the third stage, produced in the lower portions of the intestine. All of these changes were especially distinct in the cecum of rabbits, rats and chickens, and in the large intestine of dogs. From histologic examination of the first stage, a degeneration and necrosis of the epithelial cells were revealed, containing very scanty and irregular black pigment (iron reaction negative), especially in the ends of the villi. In later stages a moderate hemorrhage was found, with necrosis of the mucous membrane and with intensive infiltration of round cells in the submucosa or in the muscle strata.

Findings in *very young animals* (13 rabbits ranging from 3 to 20 days old, 2 dogs a few days old, 10 chickens ranging from 3 to 11 days old): A diffuse catarrh in all regions, especially distinct in the stomach and small intestine; sometimes erosions here and there; necrotic inflammation was found nowhere. Histologic examination showed a moderately diffuse degeneration of the epi-

thelial cells; here and there necrosis of the epithelium on the ends of the villi, with more or less hemorrhage and a very slight infiltration of round cells.

In the case of rabbits 3 weeks of age there was a moderate hemorrhagic inflammation in the cecum. The findings in animals 2 or 3 months old were similar to those in grown animals.

Experimenting with *artificial sulphuration*, as with the bismuth cases, a necrotic inflammation in a part of the fundus ventriculi and in the cecum of grown rabbits was found, while a catarrh and erosion in all regions of the intestine were observed in very young rabbits.

It is well recognized that intoxication by mercury produces a diphtheritic inflammation in the lower regions of the intestinal tract. The production of this inflammation, however, is variously explained; for instance, it was thought to be produced by the excretion of the metal (Heilborn[2]); or it might be produced secondarily to a disturbed circulation (Kaufmann[3]); or possibly it was caused by secondary bacterial infection (Virchow[4]). Sawai[1] first proved this inflammation to be caused by the sulphuration of the excreting metals. From my findings, let me say, the excretion of mercury in the intestinal tract of grown animals is most distinct in the lower portions, while in very young animals it is moderately diffuse.

3. *Silver Experiment.*—In *grown animals* (3 rabbits, 4 white rats, 1 chicken, and 2 pigeons) the capillary walls in the mucosa of the intestinal tract were, here and there, black in color, especially distinct in the lower regions. The migratory cells were moderately filled with black pigments. In the epithelial cells, however, pigment was seldom found in any region. A catarrh or very slight hemorrhage was seen in the mucosa of all regions.

In three *very young rabbits* the findings were of a little different character. The coloring of the capillary walls and the phagocytosis of the migratory cells were only slight in degree, and the excreting pigment in the epithelium was found especially in the stomach and small intestine, even though slight.

4. *Arsenic Experiment.*—In *grown animals* (7 rabbits, 7 white rats, 1 dog, 3 chickens, and 3 pigeons), a hemorrhagic inflammation was noticed in the small intestine, especially distinct in the middle portions, and more or less catarrhal changes in the other regions of the intestine. Histologic examination showed degeneration, necrosis, and desquamation of the epithelial cells, with moderate hemorrhage and infiltration of round cells.

In four *very young* rabbits ranging from 4 to 9 days old, a diffuse catarrh in the intestinal tract, especially distinct in the stomach and small intestine, was in most cases produced. In the histologic examination, swelling and degeneration of the epithelium, hyperemia and very slight infiltration of round cells were found.

5. *Chromium Experiment.*—In *grown animals* (5 rabbits, 3 white rats, 2 dogs, 5 chickens, and 2 pigeons) a catarrhal inflammation was produced which extended from the lower regions to the small intestine. In the case of dogs, erosions and partly necrotic inflammations were found in the large intestine, but these were slighter than in the case of mercury poisoning. Histologic examination showed degeneration, necrosis, and some desquamation of the epithelial cells, with infiltration of round cells, and slight hemorrhage, except in cases of necrotic inflammation.

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2. Heilborn: Arch. f. exper. Path. u. Pharmakol., 1878, **8.**
3. Kaufmann: Virchow's Arch. f. path. Anat., 1889, **117,** 227.
4. Virchow: Deutsch. med. Wchnschr., 1887, p. 1047.

In *very young animals* (five rabbits ranging from 4 to 18 days old, and a dog a few days old) a diffuse catarrh was found, especially distinct in the stomach and small intestine, and sometimes accompanied with miliary hemorrhages and erosions. Histologic examination showed a diffuse swelling and degeneration of the epithelium, edema, slight hemorrhage, and very slight infiltration of round cells.

6. *Cantharides Experiment.*—In *grown animals* (5 rabbits, 4 white rats, 1 dog, 3 chickens, and 2 pigeons), a catarrhal enteritis was produced which was most intensive in the small intestine, and was distinguished by its desquamative nature. Histologic examination showed a diffuse swelling, degeneration, necrosis and intensive desquamation of the epithelial cells, together with intensive exudation and infiltration of round cells; but hemorrhages were rare and slight.

In three *very young rabbits* ranging from 3 to 9 days old, and a dog a few days old, a diffuse catarrh was found in all regions, especially intense in the stomach and small intestine. Histologic examination showed a diffuse retrogressive alteration of the epithelium, edema, and a very sight infiltration in the membrana propria and submucosa.

7. *Ricin Experiment.*—In *grown animals* (9 rabbits, 1 dog, 7 white rats, 6 chickens, and 3 pigeons) were found catarrhal inflammation in the first stage, hemorrhagic inflammation of Peyer's patches in the second stage, diffuse hemorrhagic inflammation in the small intestine in the third stage, especially distinct in rabbits and rats. In other regions of the intestine more or less catarrh was observed, and some hemorrhage. Histologic examination showed intensive hyperemia and hemorrhage, with infiltration of round cells in the mucosa of the small intestine. In the lymph nodes, especially in Peyer's patches, diffuse hemorrhage was found, associated with swelling, phagocytosis, and degeneration of the reticuloendothelial cells of the follicles. The epithelium of the mucosa was degenerated, necrosed, and desquamated.

In the case of *very young animals* (11 rabbits ranging from 3 to 30 days old, 3 dogs ranging from 2 to 30 days old, and 2 chickens 3 days old) a diffuse catarrh was produced, extending throughout all regions of the intestinal tract; sometimes more or less hemorrhage was found in various regions, but never distinctly located in the small intestine. Histologic examination showed slight infiltration and hemorrhage, with swelling or some degeneration of the epithelium.

In cases of rabbits 3 months old the findings were similar to those of grown animals.

Kusama[5] also wrote as the result of experiment that ricin produced a hemorrhagic inflammation in the intestinal mucosa.

8. *Snake Poison Experiment.*—Findings in *grown animals* (9 rabbits, 4 white rats, 2 dogs, 2 chickens, and 3 pigeons) showed catarrhal inflammation in the first stage, hemorrhagic inflammation in the second stage, and necrotic inflammation in the third stage in the stomach mucosa. Necrotic inflammation was especially located in the middle third of the larger curvature, *at the same place where metals such as bismuth and mercury were sometimes noticeably excreted.* In the convalescent stage the mucous membrane of this part became cicatrized. Catarrhal changes were also observed to some extent in other regions, gradually diminishing as one approached the lower portions of the intestinal tract.

Histologic examination in the first stage showed an intensive swelling and some degeneration of the epithelial cells in the mucosa of the stomach, together with a moderate hyperemia. In the second stage, a moderate hemorrhage was found, principally in the propria. In the third stage, a diffuse necrosis of the mucous membrane was observed, together with intensive hemorrhage and infiltration in the submucosa. And in the convalescent stage, an intensive proliferation of the connective tissue was produced, with retrogressive altera-

5. Kusama: Zieglers Beitr. z. path. Anat. u. z. Allg. Path., 1913, **55**, 459.

tion of the epithelial cells. In other regions of the intestine a trace of the foregoing changes only was seen, but nowhere was necrotic inflammation found.

Findings in eight *very young rabbits* ranging from 4 to 9 days old showed a diffuse, intensive, catarrhal inflammation, especially in the stomach and small intestine, together with a slight hemorrhage and some erosions; necrotic inflammation was nowhere found. Histologic examination showed swelling, degeneration, some desquamation of the epithelium, hyperemia, and, rarely, a slight hemorrhage.

Two animals were fed starch paste, *that the content of the stomach might be analogous with that of grown animals,* but no new results were found.

9. *Dysentery Toxin Experiment.*—Findings in *grown animals* (10 rabbits, 4 white rats, 2 dogs, 3 chickens, and 3 pigeons): First stage, catarrhal inflammation; second stage, hemorrhagic inflammation; third stage, necrotic inflammation, and in later stages, a cicatrization in the cecum (especially of rabbits) and large intestine (especially of dogs). These results are similar to those of Flexner[6] and Doerr.[7] In rats and pigeons a hemorrhagic inflammation was observed also in part of the small intestine.

Histologic examination: In the earlier stages, the epithelial cells were swollen in the lower portions; their nuclei seemed vesicular, or they were degenerated, showing the figures of karyolysis, or they were necrotic and desquamated. The membrana propria and submucosa were intensely edematous, with an infiltration of round cells and some bleeding. The hemorrhage and infiltration were most intensive in the area of necrotic inflammation, often extending into the muscle strata. In the protracted course of the intoxication some proliferation of the connective tissue in the mucous membrane was seen. In the lymph follicles of the vermiform process was found a swelling, a degeneration, and at times a necrosis of the reticulo-endothelial cells.

Findings in *very young animals* (11 rabbits ranging from 3 to 30 days old, 3 dogs, ranging from a few days of age to 2 weeks, and 7 chickens ranging from 5 to 11 days old): In cases examined in less than three weeks after birth the most distinctive changes were catarrhal or hemorrhagic inflammation in the stomach and small intestine, with more or less bleeding in the lumen. Of course, in the cecum and large intestine certain slighter catarrhal changes and some erosions were found; nowhere was a typical necrotic dysenteric inflammation found.

Histologic examination: The epithelial cells were diffuse and moderately swollen; their cytoplasm became distinctly granular and often showed vacuolation; their nuclei were vesicular or showed some figures of karyolysis, and occasionally the epithelium was necrotic and desquamated, especially on the ends of the villi. In the section stained for fat, a moderately diffuse fatty degeneration of the epithelium was seen, not only near the ends of the villi, but often on the glandular parts. The membrana propria and submucosa were more or less edematous, with more or less hemorrhage and slight infiltration of round cells. These findings were especially distinct in the small intestine and stomach. Peyers patches were often swollen, lymph follicles were often proliferated in some degree, and sometimes there was bleeding.

In animals over 2 months of age the findings were similar to those in grown animals.

10. *Paratyphoid B Toxin Experiment.*—In *grown animals* (6 rabbits, 5 rats, a dog, 2 chickens, and a pigeon) catarrhal or hemorrhagic inflammation was produced, which was usually distinct in the cecum and large intestine and slight in the small intestine. In the last stage of the poisoning a necrotic inflammation was found in the cecum of rabbits and rats. In comparison with cases of dysentery toxemia, this necrotic inflammation was of a more localized character and of smaller area. Histologic examination showed that the necrosis

6. Flexner: Jour. exper. Med., 1906, **8,** 514.
7. Doerr: Ergebn. d. allg. Path. u. path. Anat., 1912, **16,** 122.

of the mucous membrane was of a superficial character, but hemorrhage was moderately distinct. In the earlier stages the swelling and degeneration of the epithelial cells were found, with more or less infiltration of round cells.

The lymph follicles in the intestinal tract were more or less swollen. Histologic examination showed a moderate proliferation of the reticulo-endothelial cells in the follicles, especially distinct in the vermiform process and the ileocecal patch; necrosis of the proliferated cells was nowhere observed, as is found in the case of typhoid-toxin poisoning. The lymph cells, also sometimes were more or less proliferated.

In *very young animals* (3 rabbits ranging from 3 to 10 days old, a dog 5 days old, and 6 chickens ranging from 3 to 13 days old) catarrhal inflammation was chiefly found, together with slight hemorrhage, at times, in all regions, and especially distinct in the stomach and small intestine. Moderately diffuse swelling and degeneration of the epithelial cells, slight hemorrhage, and a slight emigration of round cells were seen histologically.

In the lymph follicles a proliferation of the lymph cells was seen in the foreground, while the reticulo-endothelial cells were not so distinctly proliferated.

An Experiment with Typhoid-Toxin.—In the case of a *grown rabbit,* which was poisoned with typhoid toxin, was observed an intensive proliferation and a diffuse necrosis of the reticulo-endothelial cells in the vermiform process and ileocecal patch—typical typhoid changes—while in two cases of *very young rabbits,* under the same conditions, no such typical typhoid changes were produced. In other regions of the intestine nothing more was seen than a catarrh of the mucosa.

From the foregoing findings we may conclude that the paratyphoid B toxin produces changes closely related to but slighter than those of experimental dysentery in the lower portions of the intestine, combined with changes nearly related to those of experimental typhoid in the lymph follicles of the intestine.

Pathologic-anatomic observations on paratyphoid B disease, as far as I know, has not been reported on extensively until recently. Huebschmann[8] wrote that this disease produced, in most cases, slight dysenteric changes in the large intestine, without typical typhoid changes anywhere. Ring[9] and Buday[10] have seen miliary ulcers in Peyer's patches and in the mucosa of the small intestine; Saltykow[11] has found typhoid changes in several cases and dysenteric changes in others.

From the findings of these men it is evident that the paratyphoid B disease should produce changes closely related to those of dysentery, combined with changes nearly related to those of typhoid, justifying the conclusion that the changes which result from experimental poisoning with paratyphoid B-toxin are caused by the excretion of this toxin into the intestinal tract.

11. *Cholera Toxin Experiment.*—In cases of *grown animals* (8 rabbits, 2 rats, a dog, 3 chickens, and 2 pigeons) catarrhal inflammation was chiefly found in the small intestine (together with some hemorrhage) distinguished especially

8. Huebschmann: Ziegler's Beitr. z. path. Anat. u. z. allg. Path., 1913, **56,** 514.
9. Rings: Med. Klin., 1907, p. 1007.
10. Buday: Centralbl. f. Bact., Part 1, Orgl., 1911, **60,** 449.
11. Saltykow: Virchows Arch. f. path. Anat., 1913, **211,** 467.

by its desquamative nature. Histologic examination distinctly showed desquamation of the superficial epithelium and a moderate swelling of the deep epithelium, and also a moderate infiltration of round cells.

In cases of *very young animals* (3 rabbits ranging from 3 to 5 days old and 5 chickens from 3 to 30 days old) the changes in the stomach mucous membrane were also distinct, so that the stomach contents were often mixed with blood, and in the stomach and small intestine the alteration of the epithelial cells was diffuse, while the infiltration of round cells was always very slight.

CONCLUSION AND DISCUSSION

1. *Excretion of Poisons in the Gastro-Intestinal Tract.*—The mucous membrane of the gastro-intestinal tract of the animals poisoned by the injection of the poisons mentioned usually showed certain changes. These changes were not of the same degree in all regions, but acted most violently on a certain region; for example, on the stomach, small intestine, cecum or large intestine, depending on the kind of poison used; even though some differences were found, these were caused by the varied forms of intestinal tracts, or by the peculiar predisposition of different animals.

A problem arises, How far does the chemical action of the intestinal content (combined with excreted poisons) influence these principal changes of the intestine? No definite answer can be determined by experimental findings, but I shall state several important facts concerning this problem.

The principal change in grown animals caused by bismuth and mercury was most distinct in the cecum of rabbits; in the case of rats it was moderately localized to about the middle portion on the side toward the mesenteric fixation of the cecum; while in dogs it was most distinct in the large intestine.

The dysenteric changes in the case of rats usually extend to the middle portions of the small intestine, while it was chiefly localized in the cecum of rabbits and in the large intestine of dogs.

In the mucosa of the vermiform process of rabbits, no such characteristic changes were produced as in the mucosa of the cecum, when poisoned with bismuth, mercury, dysentery, and paratyphoid B toxins.

The changes in the small intestine in most cases were most distinct in the middle portions.

The changes in the stomach were localized to a certain part of the fundus.

From these facts we may conclude that the poisons injected were excreted most distinctly in certain regions of the intestinal tract, and this was fundamentally responsible for the specific localization of the principal changes in the intestine, even though the chemical action of the intestinal content might take a part in causing the formation of these changes.

From the data obtained we can divide the poisons into five groups according to the principal region of their excretion in the intestinal tract.

Group 1. Poisons which are most distinctly excreted in the stomach mucosa; for example, snake poison.

Group 2. Poisons which are most distinctly excreted in the small intestine; for example, arsenic, ricin, cantharidin and cholera toxin.

Group 3. Poisons which are most distinctly excreted in the lower portions (the cecum and large intestine); for example, bismuth, mercury and dysentery toxin.

Group 4. Poisons which are most distinctly excreted in the lower regions, with a noticeable excretion in the small intestine; for example, chromium and paratyphoid B toxin.

Group 5. Poisons which are not distinctly excreted in any region of the intestine; for example, silver.

The excretion of poisons by the mucous membrane of the gastrointestinal tract seems to arise in three ways: (1) secretion by epithelial cells; (2) phagocytosis of the migratory cells; (3) phagocytosis of the reticulo-endothelial cells in the lymph follicles of the intestine.

These phenomena were recognized microscopically in cases of bismuth tested animals. In other cases, also, we noticed an alteration of the epithelium, an activity of migratory cells, and a swelling of reticulo-endothelial cells. In most cases, however, excretion by the epithelium appears to be the most important factor.

2. *Enteritis by Excretion.*—Enteritis by excretion, as I will call it, is an inflammation of the mucous membrane produced by the excretion of poisonous substances into the intestinal tract. The anatomic and histologic changes of this enteritis depend on the quality of the poisons. Enteritis by excretion of mercury, which has an intensively corrosive effect, is of a hemorrhagic and necrotic character; while the enteritis by excretion of bismuth, which has a milder effect, usually shows no such intensely destructive tendency. The dysentery toxin produces a necrotic affection in the lower regions of the intestine, while the excretion of the cholera toxin results in a desquamative catarrh of the small intestine.

Or, we may say that the changes of this enteritis depend on whether the excreting poison affects most intensively the epithelium, the vessels, or the connective tissue.

From data obtained we may divide poisons into three groups, according to the nature of enteritis produced by their excretion in the intestinal tract:

1. Catarrhal poisons: silver, bismuth, chromium, cantharidin and cholera toxin.

2. Hemorrhagic poisons: arsenic and ricin.

3. Necrotic poisons: mercury, snake poison, paratyphoid B, and dysentery toxin.

Of course, the catarrhal poisons may result in more or less hemorrhage, as in like manner, hemorrhagic poisons may produce, secondarily, partial necrosis, as in the first stage of poisoning by hemor-

rhagic or necrotic poisons we only observed a catarrh. Therefore, the changes of this enteritis also depend on the doses used and the stage of the poisoning. Moreover, the animals may show a peculiar predisposition, in some degree, toward each poison.

In all stages of poisoning, however, this enteritis showed a character which was specific to a certain degree for each kind of poison, but it was especially specific in the principal region of intestinal excretion.

In the histologic examination of this enteritis there were two factors noteworthy, namely, a destructive process and a reactive process, especially distinct in the principal regions of intestinal excretion. The reason why a poison elects a certain region of intestinal excretion, is not clearly explained. It is interesting that the principal changes found in experimental enteritis by excretion of dysentery, paratyphoid B, typhoid and cholera toxins are similar to those which we usually find in necropsies after dysentery, paratyphoid B, typhoid, and cholera. Therefore, each of these bacterial toxins will find a principal region for intestinal excretion, which region is the place of predilection of these bacterial infections. Hence, we may say that every poison will have the greatest affinity for its principal region of intestinal excretion.

3. *Compensational Excretion in the Intestinal Tract.*—In several groups of chickens and rats the cecum was extirpated; then the animals were poisoned with bismuth and mercury. The most distinctive changes were observed in the large intestine and in the middle portions of the small intestine. In the cases of pigeons, which anatomically have the most rudimentary cecum, the most intensive changes were usually produced in the large intestine and in part of the last loop of the small intestine, when they were poisoned with bismuth, mercury, and dysentery toxin. These changes in the small intestine must be due to the result of a compensational excretion of poisons.

Also, in cases of most acute poisoning of grown animals by all the poisons used in our experiments, the changes produced were found to be, to a certain degree, of a more diffuse character than in cases of subacute poisonings. This result must be also due to a compensational excretion in all regions of the intestinal tract, and it was more or less closely related to the general findings of very young animals.

4. *Excretion of Poisons in the Intestinal Tract of Very Young Animals.*—The method of excretion of bismuth injected in very young animals was so characteristic that the excreted bismuth granules were found microscopically to be moderately diffused throughout all the intestinal tract; and not alone in the surface epithelium, but also in the deep epithelium; while in the case of grown animals the metal was

excreted, principally and in more concentrated form, from the epithelium on the ends of the villi in the cecum and large intestine.

In all stages of experimental poisoning with mercury, ricin, snake poison, dysentery and paratyphoid B toxins, the intestinal mucosa of the very young animals showed neither typical necrotic nor typical hemorrhagic inflammation in the described region, as is usually the case in grown animals, but an enteritis of a diffuse, and, in most cases, of a catarrhal nature.

By histologic examination a diffuse epithelial alteration (swelling and degeneration showing the figures of karyolysis, and often a diffuse fatty degeneration) was always found; a slight hemorrhage might also be found here and there, while the reactive infiltration of the round cells in the mucous membrane was usually very slight. In the lymph follicles of the intestinal tract, however, was often seen a swelling, especially in cases with bacterial toxins, and a proliferation of lymph cells.

From data obtained in regard to the excretion of poisons in the intestinal tract of very young animals, three characteristics are revealed:

1. The excretion takes place diffusely and is consequently thinned.

2. The enteritis by excretion is anatomically of a diffuse character and less specific to every poison offered.

3. The destructive process is relatively distinct in regard to the histologic findings, showing a diffuse epithelial alteration, while the reactive process is slighter in comparison with findings in grown animals.

The imperfect development of the tissue and the imperfect differentiation in the function of the mucosa ought to be responsible for these characteristics.

There are several reasons to support this opinion. From an anatomic standpoint, the intestinal tract of very young normal animals offers something of especial interest. The important points are that the formation of the glands in the intestinal mucosa is not yet perfected and the mucosa is poorer in connective tissue and wandering cells, while it is richer in vessels, in comparison with that of grown animals.

In cases of young animals ranging from 3 weeks (at which time the animals usually begin to take some food besides milk from the mother animals) to about 2 months of age, the physiologic structure of the intestinal mucosa gradually approaches that of grown animals; in like manner, I often saw, in experimenting with young animals of these ages, that the enteritis produced gradually approaches the findings in grown animals.

5. *Comparative Observations of the Pathologic-Anatomic Lesions of Enteritis in Infants with Enteritis by Excretion in Very Young Animals.*—In most cases of nutritional disturbance or infectious enteritis of infants the pathologic-anatomic lesions are of a diffuse nature

in all regions of the intestinal tract, often particularly distinguished by diffuse alterations of the epithelial cells.

Infectious diseases of the intestine of infants, such as dysentery and typhoid, often have atypical clinical signs, and pathologic-anatomic lesions often appear less severe in degree than in adults, but with atypical localization, inclined to be of a diffuse nature.

The foregoing characteristics in infants are closely related to the figures found in the enteritis by excretion of very young animals, and must be due to the imperfection of the development and function of the intestinal mucosa.

In regard to the imperfect development of the tissue of the intestinal tract in infants, there are statements by Baginsky[12] and Gundobin.[13] I have examined the intestines of several infants and about forty infants dead of enteritis. The results will be made known in the future.

By the same method of reasoning it may also be explained that the so-called "enteritis by parenteral infection," which may be an enteritis by excretion, usually causes much severer disturbances in infants than in adults.

The swelling and proliferation of the lymph follicles which are often seen in nutritional disturbances or enteritis in infants, may be caused, to a certain degree, by poisonous substances produced in the intestine, according to the results of my experiments. This view is in accordance with that of Finkelstein.[14]

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12. Baginsky: Virchows Arch. f. path. Anat., 1882, **89,** 64.
13. Gundobin: Die besonderheiten des Kindesalters, 1912, pp. 263 and 303.
14. Finkelstein: Lehrbuch der Sauglingskrankheiten, 1912, **3.**

A NEW APPARATUS FOR PUNCTURE OF THE SUPERIOR LONGITUDINAL SINUS*

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The value of the superior longitudinal sinus as a route for venipuncture for therapeutic and diagnostic purposes in young infants has recently been established. This procedure is now generally accepted as safe and practical. Tobler[1] was the first to study the anatomic relations of the sinus for the purpose of using it as means of obtaining blood and administering fluids. He showed that the puncture is best made in the midline at the posterior angle of the fontanel, where the sinus has its greatest depth. Tobler's measurements show that the distance from the skin to the lumen of the vein is from 2 to 5 mm., according to age and individual variations, and that the depth of the sinus at the posterior angle of the fontanel varies from 4 to 7 mm.

While sinus punctures are now made in practically every hospital for infants whenever venipuncture is necessary, a suitable apparatus combining the advantages of simplicity, cheapness and easy applicability has not been described. Helmholz[2] has devised an apparatus for direct transfusion into the sinus. It is a combination of the Unger apparatus with a device which steadies the baby's head and supports an ordinary needle introduced obliquely into the posterior angle of the fontanel. The apparatus has to be attached to an operating table and is rather complicated and cumbersome. The needle is introduced through a guide fastened to the child's head by adhesive plaster and set at a definite angle with the scalp. No provision is made, however, for guarding against penetration of the dura, as the needle is fastened in place only after the puncture is made.

Brown and Smith[3] used an ordinary needle, with a short bevel, on which was mounted a ridge of solder about 2 mm. thick, a quarter of an inch from the tip of the needle. This did not allow for adjustability. Several needles with various lengths beyond the guard would have to be kept on hand, which, through repeated sharpening, would soon become too short for use. Furthermore, the needle is difficult to keep steady when held between the thumb and forefinger against the child's head.

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* Submitted for publication Aug. 15, 1918.
* From the Babies' Hospital.

1. Tobler: Monatschr. f. Kinderh., 1915, **13,** 384.
2. Helmholz, H. F.: Am. Jour. Dis. Child., 1915, **10,** 194.
3. Brown, A., and Smith, G. E.: Am. Jour. Dis. Child., 1917, **13,** 501.

A simple apparatus has been devised by the writer which we have been using in the wards of the Babies' Hospital with satisfaction. It is adjustable to any desired length; it can be held securely in place despite movements of the infant's head, and it provides against puncture of the dura or injury to the brain cortex.

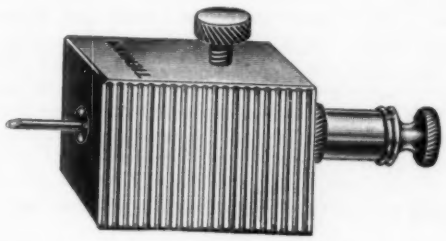

Fig. 1.—Author's apparatus.

The apparatus consists of an 18-gage needle with an obturator, a cylindrical sleeve and an aluminum block (Fig. 1). The barrel of the needle is 4 cm. long and has a bevel of 45 degrees. The hub is threaded and made to screw into the cylinder, which is 3 cm. long and 4 mm. in diameter, thus allowing 1 cm. of free needle to project beyond it. The cylinder with its contained needle fits into a bore through the middle of the aluminum block. The latter carries a set screw which secures the cylinder, setting the needle at any desired length beyond the block. A gage on the cylinder indicates the number of millimeters which the needle projects. Two opposite sides of the block are corrugated in order to insure a firm hold.

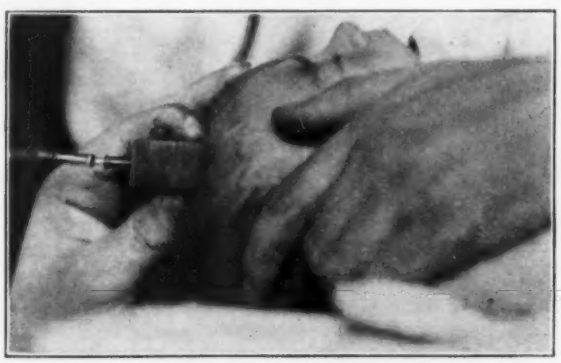

Fig. 2.—Method of using apparatus.

On examining Tobler's measurements, it will be seen that the distance from the skin to the apex of the sinus at the posterior angle of the fontanel will vary from 6 to 12 mm. in infants of different ages. It would seem, then, from these figures, that inserting the needle to the depth of 4 mm. in a young infant and 6 mm. in an older one, would bring the needle well within the lumen of the sinus without the

slightest danger of either injuring its walls or penetrating beyond them into the subdural space. In our experience we have found this to be the case.

The needle is adjusted at the desired distance and secured by the set screw. The sinus is entered at the posterior angle of the anterior fontanel at right angles to the long axis of the skull. When the needle is in place in the sinus the aluminum block rests against the child's head and prevents movements of the needle in any direction (Fig. 2). The crying of the infant raises the pressure in the sinus, so that when the obturator is removed the blood begins to flow. A syringe or gravity apparatus, as the case may be, is quickly attached to the hub and the procedure carried out in the usual manner. It is not necessary to replace the obturator before removing the needle. The reason for making the apparatus with an obturator is to obviate the occasional possibility of blocking the needle during its passage through the skin. This has occurred with us on two occasions while using an ordinary needle for sinus puncture.

The apparatus can be manufactured at a small cost; it is extremely simple, compact and easily manipulated; and it can be employed without any of the dangers or disadvantages presented by the use of ordinary needles in this work.

135 East Fifty-Fifth Street.

TERTIAN MALARIA IN TWO INFANTS UNDER TWO MONTHS OF AGE*

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It is not unusual for practitioners in our section to see malaria in its various expressions among children and adults, but it is unusual to note this particular malady in infants under 6 months of age. Medical literature has not given us many references concerning the signs and symptoms referable to malaria in the very young, and the writer presents these two cases in the belief that the manifestations expressed in these cases may be of sufficient interest to justify their publication.

CASE 1.—Lillian P., aged 2 months, was brought to me on account of fever. She was an only child. Her father is living and healthy, but her mother died at the time of her birth from puerperal fever. Her feeding history has been a most irregular one, several of the proprietary foods having been given with but little success. At the time of admission a condensed milk formula was being given. She was admitted to Touro Infirmary, July 15, 1918.

Physical Examination.—A frail infant, poorly nourished and developed, possessing marked cachexia. The mucous membranes were extremely pale and the musculature flabby. She apparently was very listless and did not respond as would the usual child when a physical examination is made. Her reflexes were remarkably diminished. The findings in the chest and abdomen were negative, the exception being that the spleen was markedly enlarged, the lower border approaching the upper border of the pelvis. On palpation the spleen was moderately hard and not particularly of the type one would expect in malaria.

Laboratory Findings.—Urine, negative; stools, constipated movements, well digested; smear (vaginal), negative; blood, hemoglobin, 25 per cent.; total red count, 2,000,000; total white count, 15,000; tertian plasmodium found in large numbers.

Treatment.—Quinin was given at intervals of two hours, 0.4 grain, in three doses, the last dose being given two hours before the expected rise in temperature. The infant was discharged from the hospital, July 22, and up to this writing has had no recurrence of temperature elevation and has made a steady gain in weight.

CASE 2.—Aug. 6, 1918, a female child was admitted to the Touro Infirmary on account of continued fever which she had had for four weeks. The temperature did not respond to the usual medication and the physician was at a loss to account for this prolonged elevation of temperature.

Physical Examination.—A desperately sick infant, appearing in a state of profound stupor; aged 7 weeks; marked cachexia; mucous membranes extremely pale; the musculature lacked tonicity and from the appearance of the skin this baby undoubtedly had lost in weight. Other than a murmur elicited from the base of the heart and a typical malarial spleen, much larger than in the first case, the infant did not present any clinical manifestations that would be of service in making a diagnosis.

* Submitted for publication Aug. 21, 1918.

Laboratory Findings.—Blood: hemoglobin, 25 per cent.; total red count, 2,600,000; total white count, 13,750; tertian malarial plasmodium found in large numbers; vaginal smear, negative.

Treatment.—After having been treated for five days, the infant was discharged from the hospital and up to the present report had not had a recurrence of temperature elevation. It is interesting to note that the tertian plasmodium was recovered from the mother and that she gave a history of *having had malaria prior to the birth of her child.*

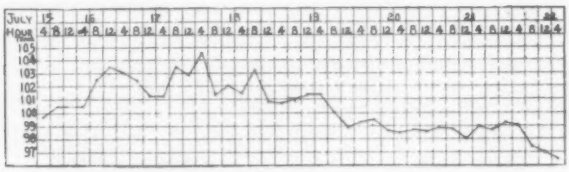

Fig. 1.—Temperature Curve in Case 1.

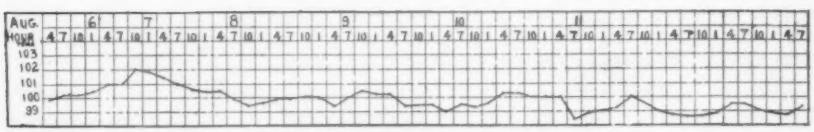

Fig. 2.—Temperature Curve in Case 2.

SUMMARY AND CONCLUSIONS

1. Tertian malaria, although uncommon in infants, does occur.

2. The common symptoms noted are: (*a*) Marked cachexia; (*b*) a corresponding marked diminution in hemoglobin and in the red blood cell count; (*c*) enlarged spleen; (*d*) prolonged temperature (atypical as compared with adults).

3. A soluble preparation of quinin, 0.4 of a grain, in three doses, was given for seven consecutive days and thereafter every other day for one month.

3529 Prytania Street.

PROGRESS IN PEDIATRICS

RECENT WORK IN ANATOMY, PHYSIOLOGY AND PATHOLOGY OF CHILDHOOD*

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(Continued from November Number)

EMBRYOLOGY AND ANATOMY

Anomalies and Malformations.—In a recent paper by the late Prof. Franklin P. Mall[63] it is calculated that the ratio of abortions to full-term births is about 1 to 4. And also that the frequency of localized anomalies in abortions is 7.5 per cent.; and that localized anomalies should be noted about twelve times as frequently as full term monsters. Studies seem to justify the conclusion that "pathologic embryos, as well as those which are normal in form, are very frequently associated with localized anomalies and that abortion usually follows as a result of serious lesions in the chorion, as well as in its environment. Should the alterations in the embryo and in the chorion be very slight, and the condition of the uterine mucous membrane, which may be expressed by the term inflammation, be overcome, the pregnancy in all probability would go to term and end in the birth of a monster or an infant presenting well recognized malformation."

Poisons produced by an inflamed uterus cannot be viewed as the sole cause, since pathologic ova occur far more commonly in tubal than in uterine pregnancy. Conditions of implantation may be a factor. Whether in the end all malformations are brought about by some simple mechanism, as alteration in the amount of oxygen or some other gas, remains to be demonstrated. It is perfectly clear that monsters are not due to germinal and hereditary causes, but are produced from normal embryos by influences which are to be sought in their environment. It may not be very far amiss to attribute these conditions to alterations in the nutrition of the embryo. Disorganization may occur, accompanied at first by cytolysis, and, as it progresses more rapidly, by histolysis, the two processes not acting with equal severity on all parts of the embryo. "When we consider the whole ovum, it is the embryo itself which is first destroyed; while within the embryo the central nervous system or the heart is the portion which is first affected."

63. Mall, F. P.: Frequency of Localized Anomalies in Human Embryos and Infants at Birth. Am. Jour. Anat., 1917, **22**, 49.

In discussing the origin of merosomatous monsters, hardly more has been stated by most authors than that there has been an arrest of development, but Mall has attempted to point out that the primary cause is in the environment of the egg, and that the arrested development is associated with the destruction of tissue. It is inconceivable to him that cyclopia should begin in an embryo after the eyes are once started in normal development; or hare-lip after the upper jaw has once been well formed; and this view is extended to include club-foot, spina bifida occulta, and other types of malformation.

Comby[64] has recently reviewed the subject of phocomelus deformity, a monster in which the hands and feet appear to be inserted immediately on the trunk, as in the seal. A number of subdivisions of the condition are recognized, and a case recently described by Mlle. Hélène Socin is given in some detail.

A. Silva Filho[65] reports the presence of a parasitic heterodelphus monster in a young child.

C. Fino[66] records the occurrence of a double monster united by the sternum. An instance of ischiopagus twins is reported by W. Salmond.[67]

Watt[68] describes with much detail a 7 months fetus that exhibited the rare deformity of complete absence of the ulna in each arm, accompanied by the still much rarer condition of monodactyly. Careful dissection of the cutaneous distribution of the median nerve in the hand of the monodactylous fetus led to the conclusion that the single finger present represented the second digit, and militated against the hypothesis that it resulted from fusion of five digits. Roentgenograms of the right and left arm are presented.

P. Lewis[69] discusses congenital absence or defects of the bones of the extremities.

Abt[70] reports a case of congenital skin defect. The baby was normal in weight and size and had been born in normal labor. It presented a defective skin area over each knee. These areas were about 1 by 1¼ inches in size, almost identical in size and shape, and seemed to be, so to speak, irregularly punched out. The base was beefy

64. Comby, J.: Phocomelous Deformity. Arch. de méd. d. enf., 1918, **21,** 33.
65. Filho, A. Silva: Heteradelphus Parasitic Monster on Young Child. Rev. med. cirug., Brazil, 1918, **26,** 30.
66. Fino, C.: Sternopagus Double Monster. Gazz. d. osp., 1917, **38,** 875.
67. Salmond, W.: Case of Ischiopagous Twins. Lancet, London, 1918, **1,** 295.
68. Watt, J. C.: Anatomy of a Seven Months' Fetus Exhibiting Bilateral Absence of the Ulna, Accompanied by Monodactyly (and also Diaphragmatic Hernia). Am. Jour. Anat., 1917, **22,** 385.
69. Lewis, P.: Congenital Absence or Defects of Bones of the Extremities. Am. Jour. Roentgenol., 1917, **4,** 431.
70. Abt, I. A.: Congenital Skin Defects. Am. Jour. Dis. Child., 1917, **14,** 113.

red and moist, and a few small foci in this red ulcerated surface showed a yellow exudate; there were indications of beginning cicatrization. These areas gradually covered over so that after five or six weeks the denuded red areas were replaced by shining, white patches, somewhat lighter in color than the surrounding skin. The healing process was complete and the baby appeared normal in every respect.

The so-called congenital skin defects have not been recorded frequently. The majority of accessible reports deal with skin defects of the scalp. They are usually circular in shape, sometimes oval or irregular, generally have clear-cut edges, and vary in size from a pinpoint upward. The first authentic case appears to have been reported by an English physician, Campbell, in 1826. Priestley reported one in 1859. Emanuel, in 1915, reported an infant with congenital absence of skin involving hands, legs, and feet. The child was stillborn; there was no evidence of syphilis.

The lesions are generally attributed to "errors in development or to inflammatory adhesions between the external layers of the skin and the amnion. At the point where the adhesion has taken place, the growth of flat epithelium is retarded." Other congenital malformations such as deficient fingers, hands, or feet, are not infrequently associated with congenital skin defects. Abt's report is accompanied by a tabulation of reported cases, by illustrations of certain of them, and by a colored plate showing the local appearance in his own case.

Litchfield[71] reports a case of "harlequin fetus." When born the child was incased from head to foot in a greatly thickened epidermis, which was cracked and seamed in many directions, but chiefly at the natural folds and wrinkles of the surface. The material formed thick, hard plates of skin, the edges of which were curling up. The natural elasticity of the skin was nowhere apparent. With careful nursing, cleanliness and anointing of the skin with white petrolatum and lanolin, the child has reached 8 years of age in a fair state of health, and is mentally bright. Her condition is one of severe ichthyosis.

Weber[72] writes on the subject of congenital hirsutes in the child.

Herbert Thoms[73] reports an instance of family prevalence of anencephaly. Ballantyne, in his series of 325 cases of monstrosity and fetal disease, noted forty-six anencephalics, an incidence of 14 per cent. In only two of the series had the mother previously given birth to an anencephalic fetus. Other instances have been noted by Lammert,

71. Litchfield, W. F.: A Case of Harlequin Fetus. Med. Jour. Australia, 1918, **1**, 494.

72. Weber, F. P.: Congenital Hirsutes in a Child. Brit. Jour. Child. Dis., 1917, **14**, 272.

73. Thoms, Herbert: Report of a Case of Family Prevalence in Anencephaly. Jour. Am. Med. Assn., 1918, **70**, 10.

Carli, and Martin. The fetus, if born alive, usually dies quickly, though Ross reported a case in which the child lived more than sixteen days. In the instances reported by Thoms, both fetuses were stillbirths. The pregnancies were not otherwise abnormal, and the Wassermann test, paternal and maternal blood, was negative, as was the microscopic examination of the placenta.

The second fetus weighed 2,150 gm. and was 48 cm. in length. It presented a typical picture of anencephaly with complete spina bifida. The head presented the well known appearance, the cranial vault and the scalp being practically absent, and this portion of the head was covered by thickened dura mater, which was dark red. The head was deeply set on the shoulders, the eyelids protruding and thickened, the forehead absent, the nose flattened, the mouth open, and the tongue protruding. The whole cerebrospinal canal from end to end was open. The vertebral arches in no place were united posteriorly, and were separated from 3 to 4 cm. most of the distance. The entire canal was covered by dura mater through which a few strands of nervous tissue could be seen. The remainder of the body was that of a well formed male child.

An instance of congenital enophthalmos is described by Lutz.[74] It appears that there have been very few publications on congenital defects of the motility of the eye. Birch-Hirschfeld (Monograph, 1917) found only 50 cases in the literature. The present article is well illustrated by photographs that show a small right eye not deflected to the right past the center line, and retracted, with diminution of the palpebral fissure in looking to the left.

McNaught[75] reports an instance of congenital occlusion of the right choana. The patient was an adult and the obstruction consisted of a long wall extending from the floor to the sphenoid, and from the posterior end of the vomer to the lateral wall. After an adequate opening had been made in the wall, it was found by testing the sense of smell with some of the essential oils that the patient could detect the odor at once on inhalation; apparently the olfactory nerves were developed.

Congenital occlusion of the choanae of the nose has been recognized since 1830, when the condition was seen postmortem by Otto. Emmert (1854) first reported the condition in the living. About one hundred cases have been reported. Cases of supposed asphyxia neonatorum may have this as a cause; such a case was reported by Richard-

74. Lutz, A.: A Case of Congenital Exophthalmos in Connection with Complete Absence of Abduction and Retraction of the Eyeball in Adduction. Arch. Ophth., 1917, **46,** 243.

75. McNaught, H. Y.: Congenital Occlusion of the Choanae of the Nose. California State Jour. Med., 1918, **16,** 193.

son in 1914. The causative factor is some departure from normal embryologic development.

Noxon Toomey[76] reports a case of congenital paramedian sinuses of the lower lip in which he summarizes the literature of the subject because its complete bibliography has heretofore been lacking in English literature.

The anomaly appears first to have been reported, in 1845, by Demarquay, who found ten cases in three families. The anomaly is usually associated with hare-lip and the lesion may consist of one or more slits leading into small saccules on the inner aspect of the lip. The sinuses may secrete mucus, particularly at meal times. Steda reviewed the literature on the subject in 1906, made a microscopic study of the lesion and concluded that the malformation arose from excessive growth through closure on both sides of the embryonal furrow of the lower lip, that is, through transformation of the lateral furrows into one canal. Huber, the histologist, arrived independently at a similar explanation. Regnier, 1913, has recently considered the subject in a monograph. Occasionally no other anomaly is present.

Toomey's case occurred in an eight months' old female infant. The baby had a complete cleft of the hard and soft palate; conditions that were successfully operated on. The more unusual anomaly is described as follows:

> Three millimeters on each side of median line of the lip and 6 mm. orally from mucocutaneous border is a sinus. The sinus mouths are not raised or inflamed and they admit nothing wider than the smallest Bowman probe. The sinuses point toward the chin and are 5 mm. deep. When squeezed they give vent to a small drop of clear mucus. Immediately orally from the mouth of each sinus is a white semilunar raised area, the cusps of which point anteriorly. Just in front of the sinus openings are hemispherically raised areas about 4 mm. in diameter.

Hughes[77] reports a case of congenital angioma of the tongue. It was seen at birth as a small swelling on the dorsum of the tongue and was diagnosed by the physician as a "birthmark." At the age of 16 years, the tumor had become as large as a marble and caused difficulty in mastication, and consequent indigestion. This condition became chronic. At the age of 26 years, the growth was the size of a large marble, reddish blue in color, well defined, with raised edges and smooth to the touch. No pain had ever been caused by it, and there had been no ulceration or hemorrhage from the growth. On pressure the growth could be reduced, but immediately filled up again when the pressure was removed. As the growth was a cavernous type of

76. Toomey, Noxon: Congenital Paramedian Sinuses of Lower Lip. Arch. Pediat., 1917, **34**, 924.

77. Hughes, F. M.: A Case of Congenital Angioma of the Tongue. Lancet, London, 1917, **2**, 749.

angioma, and benign, and the symptoms caused by it would be relieved by care and attention to the diet, it was felt best to treat the condition medically.

A case of cystic lymphangioma in the new-born is reported by R. McPherson.[78]

Ellis[79] reports a case of congenital absence of the left lung in an 8-year-old boy. The patient was of good development, but poorly nourished. He came to the hospital with a history of one week's illness, with chills and fever, and complained of pain in his knee. He died two weeks later with the following clinical diagnosis: Acute articular rheumatism; acute endocarditis; pericarditis with effusion; pleurisy with effusion.

At necropsy the left lung was found to be absent. The right lung extended well beyond the left costrochondral line and the large pericardial sac, containing 75 c.c. of fluid, occupied the remainder of the left side of the thorax. The left branch of the pulmonary artery and the left pulmonary veins were lacking. Two veins from the right lung entered the left auricle. The right lung was large, and roughly speaking, had the semblance of five lobes. The heart, other than large vegetations in the mitral leaflets, showed no noteworthy abnormality.

Complete absence of one lung is a rare condition. Ellis finds references in the literature to but eighteen of what appear to be certain cases. These he tabulates. There are five others that may be classified as possible cases.

Of the nineteen cases of absence of one lung, thirteen were of the left and six of the right. Interference with the blood supply in early embryonic life is supposed to be the cause in all cases.

Harmer[80] notes that according to Berger's teaching the brain inclusion in an encephalocele is abnormal brain tissue, cephaloma, nonfunctionating and insensitive. Harmer amputated it in four cases, three with the knife, and once with the cautery, apparently without ill effect. The four patients recovered. The thirty-four cases comprise seven meningoceles, twenty-three myelomeningoceles, three hydroencephaloceles, and one encephalocele. The total mortality was 47 per cent.

A. P. C. Ashurst[81] reports cases of birth injuries of the shoulder.

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78. McPherson, R.: A Case of Lymphangioma in the New-Born. Bull. Lying-in Hosp., 1917, **11,** 165.

79. Ellis, A. G.: Congenital Absence of Lung; Report of Case. Am. Jour. Med. Sc., 1917, **154,** 33.

80. Harmer, T. W.: Spina Bifida and Allied Malformations—Thirty-Four Cases. Boston Med. and Surg. Jour., 1917, **177,** 353.

81. Ashurst, A. P. C.: Ann. Surg., 1918, **67,** 25.

Prince[82] reports the removal of a large cystic tumor of the uterus from a 11-hour-old baby girl. The infant presented a large hernia through which the large and small intestine could be clearly seen. There was decided lack of development of the left rectus muscle, and underneath could be felt a large tumor. This was at first thought to be a hydronephrotic kidney. At operation it was found to be a cystic tumor arising from the uterus. The left tube and ovary were missing. The endometrium appeared normal. After drainage of the cystic accumulation and replacement of the intestines the infant made an uninterrupted recovery. The writer argues for early operation on remediable congenital defects.

Novak,[83] in reporting six cases of congenital absence of uterus and vagina, states that according to Burrage the first instance of this condition was described in 1572 by Realdus Columbus. Since that time several hundred cases have been reported in the literature, most of the reports, however, being very brief, usually incomplete, and sometimes leaving doubt as to the correctness of the diagnosis. Absence of the vagina is usually associated with absence of the uterus. In the absence of menstruation, these patients may complain of definite molimina, nose bleed, or, more rarely, bloody discharge from the bowel may occur. Other anomalies may be associated, misplaced kidney being an important one. The patients all complain of nonappearance of the menstrual flow.

Bruce[84] reports delivering a multipara of an apparently normal male infant which next day presented a distended and tender abdomen and vomited bile. The child was then found to be discharging meconium through the scrotum. An opening was made into the rectum and dilated; meconium escaped. The opening was kept patent and the vomiting ceased. A month later the child was the picture of health; both orfices were open and he appearently had control over discharges at both openings. A probe when inserted through the scrotal opening passed backward and downward into the rectum.

A case of congenital umbilical hernia, ectopia vesicae and imperforate anus, is reported by F. C. Pybus.[85]

Determination of Age of Fetus.—Many reasons, purely scientific and other, make the determination of the age of the fetus of con-

82. Prince, E. M.: Anatomic Defects in the New-Born Demanding Surgical Operation; Report of a Case of Uterine Tumor. Jour. Am. Med. Assn., 1918, **70,** 1212.

83. Novak, E.: Congenital Absence of Uterus and Vagina; Report of Six Cases. Surg., Gyn. and Obst., 1917, **25,** 532.

84. Bruce, J. R.: Case of Congenital Scrotal Anus. Jour. Missouri Med. Assn., 1918, **15,** 90.

85. Pybus, F. C.: Case of Congenital Umbilical Hernia, Ectopia Vesicae, and Imperforate Anus. Brit. Jour. Child. Dis., 1917, **14,** 274.

siderable practical importance. When the date of the last menstruation or the date of a single isolated coitus is not known, estimation of the age of the fetus has usually been made on the basis of the length of the fetus. All these methods are subject to considerable error. There are many physiologic and pathologic variations and numerous factors influence the length, the weight and also the measurement of the fetus. Additional or corroborative evidence from any source is desirable.

Julius H. Hess[86] makes use of the fact that there is great regularity in the development of the centers of ossification in the fetus, and that in their consecutive appearance and large number they afford a check, as it were, one on another. The presence or absence of these is easily determined by the roentgenogram. The actual time of the anatomic appearance of the various centers is about one week ahead of the time at which they cast shadows in the roentgenogram large enough to be visible.

Basing his fact on the ronetgenographic study of a series of 55 normal fetuses whose ages had been determined from the history of menstruation and of pregnancy and from their measurements, and comparing his results with the roentgenographic studies of Bade, Hasselwander, and Lamberts, Hess concludes that the roentgenographic method of determining the age of the fetus is more reliable than determination of age based on length and other measurements, since osseous development is more regular and offers many more factors for consideration; one part of the body is a criterion of normality for the other parts, and pathology may often be readily recognized. Excellent reproductions of roentgenograms accompanied by diagrams of the fetuses of various ages, from 7 weeks onward, are included in the paper.

Heat Equilibrium.—In an article in 1915, McClure and Sauer[87] stated that studies of the relation of heat to the high infant mortality of summer have not yielded uniform results, and attributed this to neglect of an important internal factor. The external factors such as food, high outdoor and indoor temperatures, circulation of air, etc., had received most of the attention; while the most important internal factor, that is, the heat regulatory power of the infant, had been neglected.

Heat equilibrium of the organism depends on the proper balance between heat production and heat elimination. Heat production is

86. Hess, Julius H.: Diagnosis of Age of Fetus by Roentgenograms. Am. Jour. Dis. Child., 1917, **14,** 397.

87. McClure and Sauer: Clothing as a Factor in the Production of Heat Stasis. Am. Jour. Dis. Child., 1915, **9,** 490.

influenced chiefly by food (Rubner) and work (well shown by Howland). Heat elimination is dependent almost entirely on (1) radiation; (2) conduction, and (3) evaporation.

Loss by radiation and conduction is proportional to the difference between the surface temperature of the body and that of the surrounding air; it becomes zero when the difference is nil; good circulation of air aids this heat loss. Evaporation gains in importance as a means of heat loss at high temperature, coincident with the appearance of visible perspiration, and becomes the principle means of heat loss when the temperature of the surrounding air is higher than that of the body. Rubner showed the importance of heat loss by evaporation from the skin and lungs at high temperatures, and Mendelssohn found the mechanism of the heat regulation for the infant to be more efficient at high than low temperature. Studies by Helmholz[88] led him to believe that the direct effect of heat alone must be considered of relatively little importance. When heat, humidity, and diet were quite uniform, certain infants thrived, others sickened, pointing thus to some other factor allowing of greater individual variability. Such a factor he thought he found in clothing.

McClure and Sauer[87] then studied the effect of clothing on the loss of body heat in 4- to 6-weeks-old puppies. The jacketed puppies regularly showed a higher temperature than controls, the stools were frequently disturbed, and the puppies died shortly. The probable importance of overclothing as a factor in infant mortality was thus emphasized.

In a succeeding study McClure and Sauer[89] sought to furnish some more accurate data showing the influence of clothing on the heat regulatory power of the infant, particularly at high temperatures. By means of mercury thermometers and thermo-elements they determined the surface temperature of infants under different conditions of clothing and room temperature and humidity. The infants wore ordinary hospital clothing consisting usually of a thin cotton gown, double-breasted cotton undershirt, cotton hose and diaper. They were tested both clothed and five minutes after being unclothed. That clothing inhibits heat loss is well known. This is further emphasized by the fact that the temperature of the skin beneath the clothing is found to be higher than that of the same area when unclothed. McClure and Sauer tabulate their determinations, and then point out certain existing inexactnesses. Accepting their figures, however, with all due reservation, as the basis of calculation for the heat loss by

88. Helmholz: The Relation of Heat to the Morbidity and Mortality of Infants from Gastro-Intestinal Diseases. Jour. Am. Med. Assn., 1914, **63,** 1511.

89. McClure, W. B., and Sauer, L. W.: The Influence of Clothing on the Surface Temperature of Infants. Am. Jour. Dis. Child., 1915, **10,** 425.

conduction and radiation, they hold it is evident that the *clothed* infant at a moderate room temperature is capable of availing itself of only about 68 per cent. of the possible heat loss due to these factors. At high room temperature it can only avail itself of about 24 per cent. of this possible heat loss.

At a room temperature of about 88 F. (31 C.) an infant clothed in the manner described and under the conditions of their experiment, from 24 to 30 per cent. humidity, etc., approaches very closely the point where a heat loss by conduction and radiation from the body surfaces is no longer possible. The preceding study on puppies shows this state of affairs may be fraught with danger to the organism.

A third and most recent paper by the same authors[90] takes up the question of loss of body heat through evaporation, the last mentioned of the three factors, radiation, conduction, and evaporation.

Heat loss by evaporation may occur (1) from the lungs, (2) from visible perspiration, and (3) from the so-called insensible perspiration. It is data in regard to the evaporation of water from the surface of the body as insensible perspiration that McClure and Sauer now report.

Observations by Loewy[91] made on individuals whose skin was devoid of sweat glands, would seem to demonstrate that insensible perspiration is entirely independent of the sweat glands. In infancy insensible perspiration would seem to be of special importance because the sweat glands "usually do not functionate during this period of life." The determination of the insensible perspiration from the total skin surface would be desirable, but such a procedure, if at all feasible, would be very difficult indeed. McClure and Sauer present some data with regard to the loss of water from restricted areas of the skin, as determined by a method which they consider superior to that used by Galeotti and Macri.[92]

As the result of these studies they conclude that, in so far as determinations of the loss of water vapor from a restricted skin area permit of conclusions with regard to the loss of water vapor from the skin in toto, it appears that the loss of water vapor from the skin is sufficiently great to make it an important factor in the heat elimination of the infant; that the rate of ventilation has a decided effect on the amount of water vapor given off by the skin, the amount increasing with the rate of ventilation up to a certain point, when it seems that a cooling effect prevents further increase; and finally, that cotton garments (two layers of cotton material as in ordinary hospital

90. McClure, W. B., and Sauer, L. W.: Observations Regarding the Loss of Water Vapor Through the Skin in Infants. Arch. Int. Med., 1918, **21,** 428.
91. Loewy: Biochem. Ztschr., 1914, **67,** 243.
92. Galeotti and Macri: Biochem. Ztschr., 1914, **67,** 472.

clothing) do not seem to offer any obstacle to the loss of water vapor from the skin, at least not when they have been in contact with the skin for a number of hours.

Blood of Mother and Fetus.—Arthur Morse, in a study of the amino-acid nitrogen in the blood in normal and complicated pregnancies and in the new-born infant,[93] says that since the work of Fischer and Kossel teaches that the protein molecule is composed of amino-acids, we assume a close analogy exists between the synthesis of polypeptids in the laboratory and the synthesis of protein in the body. Moreover, it is natural to believe that fetal protein is built of amino-acids supplied from the mother's blood. From this standpoint peculiar interest attaches to the blood of pregnant women. One would like to know whether the maternal blood contains an unusual quantity of amino-acids during pregnancy, how the quantity of these substances in the blood of the mother compares with that in the blood of the fetus, and whether in the presence of auto-intoxications abnormal values are encountered in either circulation. With the Van Slyke method these problems may be profitably studied, though recognizing that the findings are not complete.

As the result of the study Morse concludes that the requirements of the fetus cause no increase of amino-acid nitrogen in the blood of pregnant women, over that found in normal women—8 to 14 mg. per 100 c.c. of blood; that, generally, normal values are found in pre-eclamptic toxemia and eclampsia, though exceptionally an increase appeared during convalescence; that nephritic toxemia is accompanied by normal quantities; while an excessive quantity of amino-acids occurred in cases of obscure toxemia, possibly related to acute yellow atrophy of the liver. During labor normal values were found; at its conclusion the extremes encountered were 8.1 and 13.9 mg.

In the fetal blood the amino-acid nitrogen varied between 10 and 15.8 mg. per 100 c.c. of blood. "The passage of amino-acids from mother to fetus requires the assumption of 'absorption' properties on the part of the placenta; for though the difference between the concentration of amino-acids in the two circulations is small, it is in favor of the fetus in 70 per cent. of the cases; and the higher level in that organism prevents the acceptance of simple diffusion as the explanation for the placental exchange. The 'absorption' property of the placenta probably protects the fetus against loss of material intended for construction of protein."

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93. Morse, Arthur: The Amino-Acid Nitrogen of the Blood in Cases of Normal and Complicated Pregnancy, and Also in the New-Born Infant. Bull. Johns Hopkins Hosp., 1917, **28**, 199.

Courtney and Fales,[94] in the course of some studies on nutritional infants, made a large number of determinations of the total solids of the blood, and the chlorid content of the plasma, about eighty determination of each, on sixty-seven different children, ranging from 5 weeks to 6 years of age, though most of them were under 2 years of age. These they report.

Gettler and Baker[95] carried out a very complete investigation of the blood of normal adults. Courtney and Fales find that the values for the chlorids in normal infants are almost identical with those for adults. The figures for total blood solids are lower in infants, 16 to 18, as against 20 to 25 per cent. of the total blood in adults. Lust's (1911) findings for infants correspond.

In general, all infants not in a normal condition had low blood chlorid, but, except in edema or under some unusual condition of blood concentration, the percentage of total solids in the blood of infants tended to remain constant. Children suffering from tetany showed a tendency to slightly higher total solids and higher chlorid in the plasma than was found in any of the other pathologic conditions studied. Children suffering from malnutrition and digestive disorders showed the lowest chlorid in the plasma of all those investigated. Those with nutritional edema all showed abnormally dilute blood. Infants with sclerema had nearly normal total solids in the blood, with chlorid in the plasma like that of other pathologic cases, excepting the marasmus group. There was no constant relation between the variations in total solids of the blood and those in chlorid of the plasma. Saline-hypodermoclysis seemed to affect the percentage of total solids very little, but to raise the chlorid in the plasma nearly to normal.

Teeth.—Wall[96] says that while the lesions produced by the virus of hereditary lues in the second incisors—Hutchinson's teeth—are well known, little attention is paid by clinicians to the lesions in the other permanent teeth. Dental literature contains more references to this subject; especially to be recommended is the exposition of the subject by Cavallero.[97] As Cavallero remarks, "The dental stigmata are far from being restricted to Hutchinson's teeth. We must look for them in the whole dental system, in different forms, in arrangements, alterations, and even in the absence of some of the dental

94. Courtney, A. M., and Fales, H. L.: Variation in Infants of Total Blood Solids and the Concentration of Sodium Chlorid in the Plasma. Am. Jour. Dis. Child., 1917, **14,** 202.

95. Gettler and Baker: Jour. Biol. Chem., 1916, **28,** 211.

96. Wall, Joseph S.: The Significance of Certain Dental Stigmata of Congenital Syphilis. Arch. Pediat., 1917, **34,** 765.

97. Cavallero: Dental Cosmos, 1908, **1,** 167.

units." Fournier enumerates the dental stigmata pathognomonic of hereditary syphilis as follows: (1) Hutchinson's teeth; (2) cuspal erosions of the first permanent molar; (3) multiple and systematic dystrophies of the permanent teeth.

The temporary teeth calcify during the early months of pregnancy —dentification of the first or temporary set takes place during the first three or four months of pregnancy—and they remain unaffected, or death of the fetus takes place from abortion.

The sixth year molar is the *only* member of the permanent set to dentinify during intra-uterine life; the process usually begins about the fifth or sixth month. Passini, in 1907, demonstrated *Spirochaetae pallida* in the dental sacs of a 7-month-old fetus, findings which were later confirmed by Cavallero. All the other permanent teeth calcify after birth. The incisors and canines calcify in the first few months after delivery. Which teeth are affected may depend on the time at which the infection is most active. The changes in the incisors are generally known.

The changes in the sixth year molar, which may be the only dental deformity noted, in some instances are sufficiently characteristic to be of diagnostic use. There is erosion, disintegration, of the grinding surface. "The erosion is irregular in form, the surface of the molar for a third or a half or even the whole of its surface, is pitted, excavated, and discolored. There is often a pulpy looking mass of a dirty yellow color occupying most of the face of the crown, margined as it were by the line of cusps ordinarily found on the molar teeth. The tooth has been called 'the honey-comb molar of hereditary syphilis'—it is also referred to as a 'mulberry tooth,' from its resemblance to the tip of a mulberry."

Wall thinks the occurrence of such a molar is a point of great diagnostic importance and that it is not sufficiently recognized, especially as a clue to the presence of congential syphilis before the onset of a destructive keratitis. Photographs of casts of teeth accompany the article.

Tumors and Malformed Viscera.—An instance of primary sarcoma of the lung in a boy of 5 years is reported by Parkinson.[98] The child had been discharged from a surgical ward after an operation for inguinal hernia some ten weeks before his death. Shortly afterward a cough and difficulty of breathing had appeared. The symptoms progressed until the time of his death and wasting became marked. The heart was much displaced, a small immovable nodule appeared on the second rib, and bloody pleuritic effusion developed.

98. Parkinson, J. Porter: Primary Sarcoma of the Lung. Brit. Jour. Child. Dis., 1918, **15**, 28.

At necropsy the left lung was found to be replaced by a soft, friable, hemorrhagic growth, firmly adherent to the adjacent structure. The bronchial glands were involved in the mass of the growth. Microscopically the appearance was that of a small round-celled sarcoma.

Holmes[99] reports a case of ectopia cordis in a 15-month-old infant with necropsy findings. Three forms of ectopia cordis are recognized: (1) the *cervical heart,* in which the organ is displaced high in the neck; (2) the *pectoral heart,* with fissure of the sternum; and (3) the *abdominal heart,* in which the organ has passed through a defect in the diaphragm and into the abdominal cavity. Such anomalies are not extremely rare; they are found most frequently in prematurely born fetuses.

The anatomist Haller (1706) described a case; Peacock, in 1886, cited thirteen cases and described a fourteenth. Ballantyne, in 1904, mentioned nine additional cases, and another was described by Goode in the same year. Other American cases are those reported by Holt, Jacobi, Ellis and Welch.

The description of the present case is accompanied by illustrative plates. A photograph of the patient shows a tumor mass in the epigastrium, emerging from under the costal angle and extending downward almost as far as the umbilicus. This mass at birth was "raw" and bright red; later it became covered with skin. The roentgenogram shows enlargement of the heart to the right and lines suggestive of a defect in the diaphragm. The tumor mass was firm and pulsated. It was thought during life to be a misplaced heart. At necropsy it was found to be a misplaced left ventricle. Drawings show the relation of this to the rest of the heart.

The displaced portion of the heart (left ventricle) lay below a rounded muscular mass representing a hypertrophied right ventricle, and to the left of it. From it proceeded an elongated aorta. Into it opened a small mitral valve (0.6 cm.). Communication with the right ventricle occurred through a large circular defect (1.5 cm.) in the interventricular septum. In the main the circulation of the blood followed the usual channels. The small size of the left ventricle was compensated for by the assistance given by the hypertrophied right ventricle. The whole was inclosed in an irregular pericardial sac, and lay above the diaphragm, which found attachment anteriorly at an abnormally low level.

Two cases of congenital heart defects are reported by Bergmark.[100]

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99. Holmes, James B.: Ectopia Cordis, with Report of a Case in a 15-Month-Old Infant. Johns Hopkins Hospital Rep., 1917, **18,** 287.

100. Bergmark: Svensk. Läk. Sällsk. handl., 1917, **43,** 553.

Comby[101] has reported an instance of obliteration of the inferior vena cava in a boy of 12 years.

Pyloric Stenosis.—Pylorospasm and congenital pyloric stenosis have been the subject of numerous papers during the past months. The case described by the American physician, Hezekiah Beardsley, in 1788, entitled a Case of Scirrhus in the Pylorus of an Infant, and identified in 1903 by Osler as "congenital hypertrophic stenosis of the pylorus," has been regarded as the earliest published case. The symptoms in this case appeared shortly after birth, and persisted up to the time of the child's death at 5 years of age. Foote[102] now calls attention to earlier work by an English pediatrist, George Armstrong, saying:

> While Beardsley's monograph still stands as the first separate publication dealing with pyloric spasm and stenosis, the distinction of describing and actually naming with the term we now use the symptoms and pathology of this disease of the new-born apparently belong to George Armstrong, an English pediatrist, whose work appeared in 1777, a textbook entitled "An Account of the Diseases Most Incident to Children from Their Birth to the Age of Puberty, with a Successful Method of Treating Them," etc. (London, 1777). This textbook was accorded several reprintings, and was rewritten by Buchan in 1808. Clinical postmortem findings, a typical case history, and a family history, including three similar cases, led Armstrong to conclude that a child which he had treated for "watery gripes," died from spasm of the pylorus.

The postmortem findings in the case of an infant that died about three weeks of age are described, with the statement that "it looked as if the disease had been chiefly owing to a spasm *in the pylorus*, which prevented the contents of the stomach from passing into the duodenum."

In a paper entitled "Hypertrophic Stenosis in Infants" Holt[103] presents data derived from the study of 141 cases. The clinical course and the uniform pathologic findings have convinced his colleagues as well as himself that a division of cases of pyloric stenosis of infants into spasmodic and hypertrophic types is not admissible. It is his firm belief, based on observation of these cases in the early weeks, that in all of them hypertrophy precedes the tonic spasm; but that the hypertrophy continues long after the spasm has subsided he has absolute proof.

The clinical picture presented by a case is a striking one and in the large majority of the cases seen it is remarkably uniform. If one is familiar with the condition the diagnosis of hypertrophic stenosis is in most instances easy, for the symptoms in fully four-fifths of the

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101. Comby: Arch. de méd. d. enf., 1917, **20,** 418.

102. Foote, J.: Pylorospasm and Congenital Pyloric Stenosis. Am. Jour. Dis. Child., 1918, **15,** 351.

103. Holt, L. E.: Hypertrophic Stenosis in Infants. Brit. Jour. Child. Dis., 1917, **14,** 161.

cases are classical. Enumerated in the order of their diagnostic importance Holt would place the diagnostic points in the following order: (1) the history, if obtained from a reliable mother or nurse; (2) abnormal gastric retention, observations being repeated four or five times at least; (3) peristaltic waves, not of diagnostic value unless typical; (4) the presence of a palpable tumor; (5) wasting, constipation, scanty urine, etc. The roentgen ray adds nothing of importance which cannot be discovered by a study of gastric retention and without its dangers.

Many of the milder cases recover with only medical treatment. This is described. All infants which do not improve under such treatment in the course of two or three weeks, and the more severe types in a much shorter time, should be treated surgically. Cases which come under observation after four or five weeks of vomiting and marked loss in weight are best treated by operation as soon as the diagnosis is established. Earlier operations should be abandoned for the simple external division of the circular muscular fibers proposed by Rammstedt.

Examination of the tumor in longitudinal section—it is usually about as large as a peanut or the last phalanx of the little finger, of glistening whiteness and cartilaginous hardness—shows that the hypertrophy is practically limited to the circular muscular layer. The circular fibers are much increased both in size and number; there is a true hypertrophy. In recent cases there is usually a considerable amount of edema. The longitudinal layer of muscular fibers, the submucous and mucous coats, are in most cases quite normal. Rammstedt, of Munster (1913), simply divided the circular muscular layer of the pylorus by external incision. The stomach is unopened; the operation in skilled hands is short; the patients make excellent recoveries.

Patients followed two and three years after the Rammstedt operation show no interference with health and progress; patients not operated on usually show no symptoms after the first year; yet the possibility exists that this condition may be the basis of pyloric obstruction in later life.

Ladd[104] contributes a paper on the same subject.

Ransohoff and Wooley[105] and later Rachford[106] describe the conditions found postmortem at the pylorus in a 7-month infant that died

104. Ladd, W. E.: Congenital Pyloric Stenosis. Boston Med. and Surg. Jour, 1918, **178,** 767.

105. Ransohoff, J. Louis, and Wooley, Paul G.: Operative Cure of Congenital Pyloric Stenosis; Thymic Death after Seven Months: Necropsy. Jour. Am. Med. Assn., 1917, **68,** 1543.

106. Rachford, B. K.: Pyloric Stenosis in Infancy. Arch. Pediat., 1917, **34,** 803.

suddenly four months after a Rammstedt operation had been done. The clinical history had been typical. At operation the pyloric tumor was of cartilaginous hardness, 2 cm. long and 1 cm. thick. The child's recovery after the operation had been rapid and uneventful, and at the age of 6 months it appeared in every way normal. In the middle of the seventh month the child without warning was found dead in bed. Death was attributed to the unusually large thymus (65.5 gm.).

At necropsy it was found that along the anterior surface of the pylorus there was a shallow linear depression approximately 2 cm. long, which represented the scarred site of a previous surgical incision, and in the groove and its margins were tags of old fibrous adhesions, rather tenuous in texture, which had united the pylorus to the adjacent tissues. The tumor mass had disappeared. Section showed that from the bottom of the groove to the submucosa was a band of fibrous tissue which represented a loss of muscular tissue in that region. There was no bulging, no diverticula or hernia formation. It seemed possible that the muscular layers were somewhat better developed than would be called normal, but the variation seemed too slight for weight to be attached to it. The appearance of the pylorus on section was as if a ring shaped muscle were supplied at one point with a tendon.

It appeared that the surgical incision permitted the muscular coats of the tissue to retract and the opening to gap, and that subsequently the space was filled with organized tissue, which in its fully developed state was fibrous tissue. It seemed also that during the healing of the wound the pyloric orifice became dilated and then remained so, and thereafter performed its function in an acceptable manner. There is thus afforded anatomic proof of the perfect efficiency of the Rammstedt operation.

Scott[107] reports a case of hypertrophic pyloric stenosis in a 2-year-old child with operation (Rammstedt) and death. In Scott's case there was no early history of vomiting—though he had occasionally done so during "teething," and after eating a banana. He had passed through whoopingcough. About a month before the operation he had become nauseated and had vomited following a hypodermic injection given to produce vomiting, and occasionally thereafter. There was an indefinite history of chewing firecrackers. The child presented a scaphoid abdomen with visible peristaltic waves but no palpable tumor. Six days after entrance to the hospital, it was thought advisable, as the child was getting weaker, to operate.

Operation revealed "a typical hypertrophied pylorus such as is seen in congenital pyloric stenosis." An incision (Rammstedt) showed

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107. Scott, A. J., Jr.: Hypertrophic Pyloric Stenosis in a Two-Year-Old Child; Report of a Case. Jour. Am. Med. Assn., 1918, **70**, 1918.

typical cartilaginous texture. The mucosa was accidentally nicked. The child died, with rising temperature (104.6 F.). Necropsy was not permitted.

The first reported case of pyloric stenosis in an older child is that by Beardsley (referred to in the foregoing) in which a boy showed symptoms shortly after birth, and up to his death at the age of *5 years*. In 1914, Shaw and Elting reported a case in which the patient showed symptoms from the seventh or eighth month, but was not operated on until the age of 16 months. The child lived.

Arreger reported a case in which the patient had no symptoms until he was 3 years old, and at the age of 5 years had the first marked symptoms. He was not operated on, however, until he was 6½ years old.

Grey,[108] after performing a gastric fistula on dogs as devised by Janeway, found it possible to dissect a 2 cm. long cylinder of duodenum containing the major bile duct and insert it into the posterior wall of the stomach, anastomose the ends of duodenum, and tie off the lesser pancreatic duct. The operation was readily carried out through a high right rectus or midline incision.

From the results of his experiments it appears that the presence of a considerable amount of pancreatic juice in the stomach throughout the period of digestion leads only to a moderate decrease in the acidity level of the ingesta in the late stage of digestion. Earlier in the process there is no constant alteration in the acidity level in either direction. The findings then serve not only to corroborate the views of Boldyreff, but also to demonstrate the remarkable compensatory activity of the gastric glands, under conditions which entail an unusual quantity of alkali in the stomach.

In addition the work has shown that when the larger pancreatic duct is properly transplanted into the wall of the stomach, it may remain patent for months. In animals in which the operation has been carried out, the pancreas has been found to undergo no inflammatory or other changes. The finding is regarded as evidence against the postulation of Hlava that gastric juice is probably responsible for the occurrence of certain cases of acute hemorrhagic pancreatitis.

Carcinoma.—Cosens[109] has reported an instance of primary carcinoma of the stomach in a boy.

Primary carcinoma of the liver in early life, though very rare, has been the subject of considerable study. Reported cases have been

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108. Grey, Ernest S.: The Diversion of the Pancreatic Juice from the Duodenum Into the Stomach. Its Effect on the Level of Gastric Acidity and on the Pancreas. Jour. Exper. Med., 1917, **26**, 285.

109. Cosens: Brit. Med. Jour., 1917, **1**, 649.

tabulated by Steffen and Philipp and Castle (1914). Griffith[110] analyzes the tabulations and adds twelve others from the medical literature and one of his own. This brings the total reported cases regarded by Griffith as authentic up to fifty-seven.

Of these 57 cases, 25 were in males and 26 in females. In the other instances the sex is not stated. The age of the patients varied from birth to 16 years. In two instances the age is not stated. Four were new-born, and including these there were 8 in the first three months, and 16 in all in the first year. The remainder were divided as follows: 1 to 2 years, 8; 3 years, 2; 4 years, 2; 5 years, 2; 6 years, 2; 7 years, 2; 8 years, 1; 9 years, 3; 11 years, 4; 12 years, 5; 13 years, 7; 14 years, 3; 15 years, 2; 16 years, 1.

The occurrence of so many early cases suggests the existence of a tumor of an embryonal nature; and this has been claimed for a number of them. Others are simple carcinoma; others clearly adenocarcinoma; and others, doubtless some of them carcinomatous, have been described as adenoma. The tumors may appear in various forms and may be divided (Eggel) into nodular, massive, and diffuse, the first being most common, the last rare. Metastases are not uncommon, especially to the lungs and to other parts of the liver. Operative interference has been attempted in one instance but without success. It would seem possible only in cases of a single encapsulated tumor. In view of the seriousness of the condition, any effort at removal would seem justifiable.

Griffith's case was a female infant, aged 21 months. She had previously been in good health except for some degree of anemia, which was supposed to be dependent on indigestion, and a constant tendency to constipation. The tumor had been discovered accidentally five weeks before. It was said not to have increased in size, but there was an unusual amount of veining of the walls of the thorax and of the distended abdomen; the child had lost weight and had not seemed well. Examination showed a smooth hard mass just below the costal border and midway between the midsternal and right mammary lines. It changed its position distinctly with respiration. A roentgen-ray picture seemed to show opaque bodies in the tumor mass, thus suggesting a diagnosis of teratoma connected with the kidney or with the suprarenal body. Exploratory incision revealed an orange-sized mass in the right lobe of the liver. It was covered by a dark gray, glistening, fibrous capsule, projected slightly above the surface of the liver, and extending in depth through the entire thickness of the lobe. There was no evidence of infiltration of the hepatic tissue beyond the

110. Griffith, J. P. C.: Primary Carcinoma of the Liver in Infancy and Childhood, Am. Jour. Med. Sc., 1918, **155**, 79.

capsule, nor of metastasis. Incision into the capsule showed a soft, whitish substance, much resembling in color and appearance the thymus gland or pancreas.

Histologic examination showed the tissue to consist entirely of epithelial cells without trace of liver tissue. Histologically it was a carcinoma simplex, but the presence of attempts at tube arrangement suggested that the growth was originally a cancer of the bile ducts.

Forman and Hattery[111] report a case of primary carcinoma of the liver in a boy of 3 years. Attacks of abdominal distention and pain had appeared a year earlier and grown progressively more severe. They were accompanied at times by jaundice. There was great enlargement of the liver and loss of weight. The tumor fell into the class of primary parenchymatous carcinomas of the liver, usually regarded as congenital. The specimen served to emphasize the usual absence of extra hepatic metastases in cancer of the liver.

An instance of primary carcinoma of the liver in a girl of 11 years is reported by De Saussine.[112]

Ganguli[113] writes upon Infantile Biliary Cirrhosis.

Miscellaneous Pathologic Visceral Condition.—Cockayne[114] reports a case of congenital defect of the duodenum in which bile was found both above and below the absent portion. The child was a male infant born at full term, which died on the fifth day with symptoms of intestinal obstruction. The vomitus was invariably watery and green. At the confinement the medical attendant had noticed that the liquor amnii was excessive in quantity and bright green in color. At necropsy the first part of the duodenum was found to end blindly, to be greatly distended and globular in outline, and to contain *bile-stained* fluid. The pancreatic duct entered the posterior aspect of the blind sac. There was no connection except by peritoneum with the upper blind sac and the narrow commencement of the lower part of the duodenum, not even a thin cord. The bile duct opened directly into the narrow commencement of the lower part of the duodenum. The gallbladder, liver, and ducts were normal and contained bile. Careful search failed to reveal any second bile duct, leading to the upper portion of the duodenum.

The source of the large quantity of bile-stained fluid that was vomited is the interesting feature of the case. The earliest case in which bile was found both above and below a duodenal occlusion was

111. Forman, F., and Hattery, J. S.: Primary Carcinoma of the Liver in Childhood; Report of a Case. Am. Jour. Obst., 1917, **76,** 862.

112. De Saussine: Arch. de méd. d. enf., 1917, **20,** 472.

113. Ganguli: Indian Med. Gaz., 1917, **52,** 361.

114. Cockayne, E. A.: A Case of Congenital Defect of the Duodenum in which Bile Was Found Both Above and Below the Absent Portion. Brit. Jour. Child. Dis., 1917, **15,** 188.

described by Hirschprung, who proved the presence of bile pigments in the stomach. Later cases have been reported by Wilks, Hobson, Wyss, Cordes and others. Cordes proved conclusively that in her case the common bile duct sent a small branch to the dilated duodenum above the occlusion, and gave reasons for believing that the anomaly might be present in other cases. The view of Wilks that the green fluid vomited is a secretion of the stomach may be rejected. The only reasonable alternative to Cordes' explanation is that the child may have swallowed bile-containing amniotic fluid; this seems unlikely, and in one case (Anderson's) it was specially noted that the amniotic fluid was colorless.

An instance of common cyst of the bile duct is reported by E. Waller.[115]

Eisendrath[116] reports two cases of gallstones in girls of 15 years; in the second case there was an accompanying acute cholecystitis with many calculi. Up to 1913, Khantz was only able to find the reports of fifteen cases; of these, one was in a new-born infant, five were necropsy observations, and in nine the diagnosis was confirmed, as in Eisendrath's cases, by operation. One other case has since been reported. All of the patients above 13 were girls. The stones may be numerous.

C. Martelli[117] reports a case of heart disease in a child under 3 years with ascites from the liver.

Scott[118] reports an instance of pneumococcal abscess of the liver in a 13-month-old infant, suffering from pneumonia. At necropsy there was seen a prominent bulging on the upper surface of the liver, and on opening this there was a marked outpouring of greenish pus, which on smear and culture showed pure culture of pneumococcus.

Jemma[119] reports a case of abscess of the liver in a boy of 8 years.

The subject of congenital variations in the peritoneal relations of the ascending colon, appendix, cecum, and terminal ileum has been studied by S. C. Harvey.[120]

Vander Bogert[121] reports a fourth case of congenital atresia of the small intestine. The child died four days after birth. The symptoms were those of complete obstruction. The vomitus became distinctly

115. Waller, E.: Hygiea, Stockholm, 1917, **79,** 513.

116. Eisendrath, D. N.: Gallstones in Infancy and Childhood. Ann. Surg., 1917, **66,** 557.

117. Martelli, C.: Pediatria, Naples, 1917, **25,** 401.

118. Scott: California State Med. Jour., 1918, **16,** 337.

119. Jemma: Pediatria, Naples, 1917, **25,** 129.

120. Harvey, S. C.: Congenital Variations in the Peritoneal Relations of the Ascending Colon, Cecum, Appendix and Terminal Ileum. Ann. Surg., 1918, **67,** 641.

121. Vander Bogert, Frank: Congenital Atresia of the Small Intestine, Arch. Pediat., 1918, **35,** 45.

fecal on the third day. Enemas yielded a mucus cast partially filled with a substance resembling meconium, except that it gave no reaction for bile. (Normal meconium gives a bile reaction.) Necropsy showed the obstruction to be complete, the small bowel much dilated, ending abruptly 25 inches above the ileocecal valve. There was complete absence of intestine for about ½ inch and both the small and large bowel below were poorly developed.

In the three cases earlier reported (*Arch. Pediat.*, 1916, 33, 194) the stools obtained by enema were normal in character, and the obstruction was incomplete. In the present case, as in the earlier complete case, bile appears to have been absent. Vander Bogert feels this should be regarded as conclusive proof of complete obstruction.

St. George[122] reports a case of congenital obstruction of the intestines. The patient, a girl, the second of twins, died on the seventh day after birth. A clinical diagnosis of intestinal obstruction (congenital) and hemorrhagic disease of the new-born was made. At necropsy there was found an apparently complete stricture of the duodenum about 9 cm. below the pylorus, and another in the jejunum about 20 cm. below. Microscopic section showed "complete destruction of the lumen of the duodenum and replacement by fibrous tissue in which were a number of blood vessels." Section through the second point of constriction showed a lumen which was markedly diminished in caliber and occupied by a small blood clot.

In the large necropsy service of Bellevue Hospital during the past fourteen years congenital obstruction of the intestine has been encountered twice only. References to 143 cases in the literature are appended.

Griffith[123] reports a case of idiopathic dilation of the colon, in a colored boy of 4 years. The condition had been present from his early days. His general health had always been good and while the case appeared to be a typical one of moderate degree, operative interference did not seem justifiable.

The case is reported because of the interesting roentgenogram that was obtained. Griffith distinguishes between secondary dilatation of the colon due to abnormal construction, stenosis, and the like, and Hirschprung's disease, which is in no way associated with stenosis and appears to be due to a congenital tendency for the colon to dilate. This tendency is present at birth although the actual dilatation may not show itself for some time.

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122. St. George, A. V.: Congenital Intestinal Obstruction; with the Report of a Case. Am. Jour. Dis. Child., 1918, **15**, 354.

123. Griffith, J. P. C.: A Case of Idiopathic Dilatation of the Colon, with Photograph and X-ray. Arch. Pediat., 1918, **35**, 36.

Another case with roentgenograms is reported by Baird and Kindley.[124] Their patient was a 6-year-old girl. Symptoms had appeared at 3 years. She was pale but not emaciated. The abdomen was distended. A firm mass extended from the right hypochondrium down to the pelvis and then upward to the left hypochondrium. Bismuth given at night appeared next morning in a channel extending at least partly through the mass, apparently involving the first part of the transverse colon. On moving the mass the barium could be seen to move.

Schneiderhohn (1915) found 358 cases of this condition in the literature and added four of his own. A number of others have been added. Other cases are reported by Finchietto,[125] Noble[126] and Sohmer.[127] Neugebauer (1913) advised resection and gives 56.5 per cent. of cures after immediate resection and 90 per cent. when it is done in stages. Schneiderhohn showed that of 151 cases treated medically, 25 per cent. were cured, 81 per cent. of the cured being children.

Genito-Urinary Conditions.—Many studies on the elimination of phenolsulphonephthalein in children have been made since the substance was introduced into clinical medicine in 1910. It is an ideal substance for the study of renal function, being practically nontoxic and nonirritating, readily absorbed and readily eliminated through the kidneys. Rowntree and Geraghty believe "that the capacity for picking out the molecules of phthalein from the infinitely dilute solution in the blood and passing them on into the urinary secretion in comparatively concentrative solution, is a function specific to the kidney." They have also obtained fairly conclusive proof that the cells of the tubules play the most important rôle in the excretion of the phenolsulphonephthalein, the glomeruli eliminating a very much smaller amount.

In 1912 Smith reported the findings in a small series of young patients. Fishbein, in 1913, studied a small group of children and adults. In 1915 Tileston and Comfort estimated the phenolsulphonephthalein output in several groups of patients varying in age from 6 months to 11 years. Only six of the patients were under 2 years of age. In the same year Hempelmann studied the phenolsulphonephthalein elimination in seven patients with orthostatic albuminuria,

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124. Baird, R. W., and Kindley, G. C.: Hirschsprung's Disease; Report of a Case. Southern Med. Jour., 1917, **10,** 846.

125. Finchietto, R.: Megacolon. Rev. assoc. med. argentino, 1917, **27,** 873.

126. Noble, T. B.: A Case of Megacolon. Am. Jour. Obst. and Dis. Women and Child., 1917, **76,** 946.

127. Sohmer, A. E.: Congenital Idiopathic Dilatation of the Colon. Minnesota Med., 1918, **1,** 119.

from 8 to 12 years of age; and in the next year (1916) Barker and Smith reported the findings in similar patients from 16 to 25 years of age.

Gittings and Mitchell's[128] studies in phenolsulphonephthalein elimination were made on seventy-five hospital patients varying in age from 3 weeks to 10 years; forty-four were under 1 year of age; seven from 1 to 2 years of age, and twenty-four from 2 to 10 years of age.

Their studies show that the age of the patient within this limit has no influence on the elimination; "even the youngest children and infants show about the same capacity for phenolsulphonephthalein elimination as do adults—60 per cent. for two hours and 45 for one hour, at 3 and 5 weeks." They confirm the observation that the elimination of phenolsulphonephthalein is not markedly decreased in any disease other than renal. They believe that in children a single collection exactly two hours after the injection into the lumbar muscles of 6 mg. of phenolsulphonephthalein, should be the method of choice. The necessity for continuous or repeated catheterization is thereby avoided.

The occurrence of multiple urinary calculi in a boy of 10 years is reported by Pybus.[129] Roentgen-ray examination revealed an oval calculus in the pelvic portion of the left ureter, and a second in the right kidney. The child had passed a stone per urethram three years previously. He had also been recently operated on with the extraction of a small stone which was impacted in the urethra. A few days later he passed a larger stone. The roentgenograms were then made. An accompanying cystitis was treated. A second plate made three months later showed the stones had not altered in position. They were then successfully extracted. The pyuria ceased and the child's condition, both physical and mental, underwent very great improvement. Cumston[130] shows how easily the presence of such stones may be overlooked. He then considers the technic of removing them.

Borobio,[131] Cifuentes[132] and Martin,[133] also contributed articles on renal calculi in childhood, the last mentioned reporting four cases.

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128. Gittings, J. C., and Mitchell, A. G.: The Phenolsulphonephthalein Elimination in Infants and Young Children. Am. Jour. Dis. Child., 1917, **14,** 174.

129. Pybus, F. C.: A Case of Multiple Urinary Calculi. Brit. Jour. Child. Dis., 1917, **14,** 97.

130. Cumston, C. G.: A Note on Ureteral Calculi in Childhood. Brit. Jour. Child. Dis., 1917, **14,** 94.

131. Borobio, P.: Bladder Concretions in a Child. Arch. espan. de pediat., 1918, **2,** 65.

132. Cifuentes, P.: Calculi in a Child's Kidney. Arch. Espan. d. pediat., 1918, **2,** 26.

133. Martin, A. P.: Hematuria with Kidney Stones—Four Cases. Siglo méd., 1917, **44,** 568; and Stones in the Ureters, Ibid., 1918, **55,** 124.

L. W. Hill[134] reviews the literature of the last nine years on nephritis of infancy and childhood.

Royster[135] reports the gross and microscopic findings in a case of bilateral polycystic kidneys in an 8-months-old male infant of Syrian parentage. The masses were evident at 4 weeks of age. The report is well illustrated.

An instance of congenital cystic kidney in which a tuberculous process was superimposed is reported by Woolley.[136]

Darnell[137] reports the occurrence of a mixed cell tumor of the kidney weighing 3 pounds, in a 10-months-old infant, and Kirmisson and Tretiakoff[138] report an adenosarcoma of the kidney in a girl of 6.

Mixter[139] reports an instance of sarcoma of the bladder in a young child.

Cuthbert[140] reports a metastatic glioma (neuro-epithelioma) of the right ovary in a child of 3 years.

The occurrence of typhoid gangrene in a girl of 5 years, requiring amputation, is reported by Vinache.[141]

Carcinoma of the breast in the male child is extremely rare. It has even been asserted that it does not occur. Blodgett, however, in 1897, reported a case in a boy of 12 years and Bryan reported a case in a boy of 14 years and 8 months. Simmons[142] now reports one in a boy of 13 years.

The patient was a well built, healthy boy. The family history was essentially negative. About a year earlier the boy had been struck in the right breast with a baseball bat. The blow was light and caused no immediate soreness, but soon afterward the boy noticed after exertion a burning and stinging sensation in that breast. About seven months later a swelling became noticeable and there was some tender-

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134. Hill, L. W.: Review of the Literature of the Last Nine Years on Nephritis of Infancy and Childhood. Boston Med. and Surg. Jour., 1917, **177,** 313.

135. Royster, L. T.: Polycystic Kidney in an Infant. Am. Jour. Dis. Child., 1918, **15,** 196.

136. Woolley, P. G.: A Case of Congenital Cystic Kidney in which a Tuberculous Process Was Superimposed. Jour. Lab. and Clin. Med., 1917, **3,** 55.

137. Darnell, W. E.: Mixed Cell Tumor of the Kidney Weighing Three Pounds in an Infant Ten Months Old. Am. Jour. Obst., 1917, **76,** 786.

138. Kirmisson, E., and Tretiakoff: Adenosarcoma of the Kidney in a Girl of Six. Arch. de méd. d. enf., 1917, **20,** 257.

139. Mixter, C. G.: Sarcoma of the Bladder in a Child. Ann. Surg., 1917, **65,** 628.

140. Cuthbert, Lockyer: Metastatic Glioma (Neuro-epithelioma) of the Right Ovary in a Child Aged 3 Years. Brit. Jour. Child. Dis., 1917, **14,** 199.

141. Vinache, A.: Un cas de gangiène typhoide chez un enfant. Paris méd., 1917, **24,** 402.

142. Simmons, R. R.: Adenocarcinoma of the Breast Occurring in a Boy of Thirteen. Jour. Am. Med. Assn., 1917, **68,** 1899.

ness. A diagnosis of chronic mastitis was made and the entire breast removed.

The diseased tissue was approximately 8 cm. across and about 1.5 cm. thick in the center; toward the margin the tumor was thin. The axillary glands showed no enlargement.

Microscopically there was a marked proliferation of the gland elements throughout the sections. The newly formed glands were very irregular in shape, and the epithelium was arranged in many layers. Some showed definite lumina, while in others the proliferation was so great that the lumen was entirely filled up with cancer cells. Many of the glandular structures showed an ingrowth of stroma dividing it up into many smaller divisions. There was a tendency toward the formation of grapelike clusters of these atypical glands. The ducts showed some reduplication in the layers of epithelium. The stroma was abundant but not highly cellular. The adipose tissue appeared normal except for some areas in which the cancer had grown into it. There was a tendency to degeneration of the cells which lay centrally in the atypical acini, but this had not progressed to a marked degree. The pathologic diagnosis was adenocarcinoma of the breast, of the medullary type. The case is unusual both from the standpoint of age and sex.

Ehrenfried[143] reports cases of hereditary deforming chondrodysplasia, and Opie and Allison[144] consider hypertrophic chondrodystrophy in infancy and adolescence, and Gibney[145] considers osteochondritis deformans juvenalis.

Pathology Related to the Nervous System.—Felton, Hussey and Bayne-Jones[146] in a preliminary report on the hydrogen-ion concentration of the spinal fluid, as determined by the colorimetric method, confirm the work of Besgaard, in which he states that the reaction of the cerebrospinal fluid is more acid than pH = 8.1. It is approximately that of the blood serum. Certain inaccuracies inherent in the colorimetric method of determination, as compared with the gas chain method, for determining the hydrogen-ion concentration of the spinal fluid are noted.

Reference was made last year to the studies of Schlapp and Gere[147]

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143. Ehrenfried, A.: Hereditary Deforming Chondrodysplasia; Report of Cases. Am. Jour. Orth. Surg., 1917, **15,** 463.

144. Opie, E. L., and Allison, N.: Hypertrophic Chondrodystrophy in Infancy and Adolescence. Progressive Anomaly of Osteogenesis. Jour. Med. Research, 1917, **36,** 277.

145. Gibney, V. P.: Osteochondritis Deformans Juvenalis—Perthes Disease. Med. Rec., New York, 1917, **91,** 793.

146. Felton, L. D., Hussey, R. G., and Bayne-Jones, S.: The Reaction of the Cerebrospinal Fluid. Arch. Int. Med., 1917, **19,** 1085.

147. Schlapp, M. G., and Gere, B.: Occlusion of the Aqueduct of Sylvius in Relation to Internal Hydrocephalus. Am. Jour. Dis. Child., 1917, **13,** 461.

on eight cases of internal hydrocephalus of the obstructive type, with especial reference to obliterating lesions of the aqueduct of Sylvius as the cause of the ventricular distention. Schlapp and Gere thought it not unreasonable to attribute many if not the majority of cases of congenital internal hydrocephalus to closure of the aqueduct of Sylvius through proliferation of the glia and ependymal tissues, or to the invasion of the fourth ventricle by tumors.

Dandy and Blackfan,[148] following their earlier work, divide cases of internal hydrocephalus into two apparently distinct anatomic varieties—obstructive hydrocephalus and communicating hydrocephalus. In their second study[149] they conclude that "meningitis is by far the greatest etiologic factor in both types of hydrocephalus and probably always the cause of the communicating variety. This may be either prenatal or postnatal. The meningitis may be of a very mild grade and easily overlooked. Spontaneous recovery sometimes occurs. Their material comprised twenty-six cases, fifteen of the obstructive and eleven of the communicating variety. Postmortem examinations demonstrated an obstruction in every case in which an obstruction had been shown clinically by the phenolsulphonephthalein test. "Absorption of cerebrospinal fluid is a general process, from the entire subarachnoid space, and communicating hydrocephalus results because only a fraction of the area can be utilized for absorption." Still less is available in the obstructive type.

In a symposium on hydrocephalus Elsberg[150] expressed the opinion that cases of the congenital type are entirely different in genesis and course from those of inflammatory nature. There are, according to him, three definite types: that due to obstruction, that due to diminished absorption, and that due to hypersecretion. With hypersecretion the only thing to be done is to give relief by frequent puncture and administer thyroid preparations to diminish the amount of secretion.

Of forty cases of chronic hydrocephalus studied, ten were obstructive, thirty nonobstructive, and of these last eighteen were nonobstructive with diminished absorption and hypersecretion.

Elsberg found some evidence that puncture of the corpus callosum might be worth trying in patients with obstructive hydrocephalus in whom subarachnoid absorption had been demonstrated to be below normal. In cases of hydrocephalus due to hypersecretion, lumbar

148. Dandy, W. E., and Blackfan, K. D.: Internal Hydrocephalus; an Experimental, Clinical and Pathological Study. Am. Jour. Dis. Child., 1914, **8,** 406; also Beitr. z. klin. Chir., 1914, **93,** 392.

149. Dandy, W. E., and Blackfan, K. D.: Internal Hydrocephalus. Am. Jour. Dis. Child., 1917, **14,** 424.

150. Elsberg, Charles A.: Chronic Internal Hydrocephalus. The Newer Methods for Its Recognition and Treatment. Abstr. Arch. Pediat., 1918, **24,** 851.

puncture should be done every few days (15 to 30 c.c. fluid withdrawn) and the patient should be given thyroid extract up to the physiologic limit. He felt convinced that real success could not be expected from any methods thus far devised to drain fluid into any cavities of the body (Mt. Sinai Hospital experience).

Dr. William Sharpe, on the other hand, felt that success had been attained by providing drainage through the dura into the subcutaneous areas of the scalp by means of linen thread (seventeen deaths in fifty-eight cases). From his observations at operation and at necropsy on fatal cases (fourteen out of forty-one) he believed most cases of hydrocephalus due to a form of meningitis and a blocking of the stomata of exit of the cerebrospinal fluid in the cortical veins and sinuses, and possibly lymphatics.

Dandy and Blackfan feel that the surgical treatment of internal hydrocephalus has now a definite anatomic basis and hopeful prospects.

Holt[151] reports a case of gliosarcoma in an infant of seven weeks that resembled hydrocephalus. At about 2 weeks of age the head was observed by the parents to be enlarging more rapidly than normal. A little later the veins of the scalp became very prominent. The picture of hydrocephalus was produced. The head increased greatly in size. It was somewhat asymmetrical, there being a very great prominence in the left frontal region. At 8 weeks a spontaneous rupture took place over this prominence, and a true hernia cerebri was formed. The child died two days later.

The tumor mass was found to involve practically the entire left hemisphere. It apparently grew from the floor of the lateral ventricle. Microscopic examination showed it to be a gliosarcoma.

Acute Infections.—Borries[152] reports constantly clear lumbar puncture fluid with severe "otogenous meningitis."

Davison and Miller[153] report clinical and bacteriologic observations on a case of meningitis due to a hitherto undescribed organism. The onset was sudden with convulsions, in a 6-months-old child which had been treated during the preceding three weeks for a purulent otitis media with moderate elevation of temperature. The physical examination was negative except for the double otitis media. The temperature was 102.6 F. The convulsions were promptly controlled with sedatives. Lumbar puncture yielded purulent fluid, without organisms.

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151. Holt, L. E.: Gliosarcoma in an Infant of Seven Weeks. Resembling Hydrocephalus. Am. Jour. Dis. Child., 1917, **14,** 219.

152. Borries, G. V. T.: Lumbar Puncture Fluid Constantly Sterile with Severe Otogenous Meningitis. Ugesk. f. Læger, 1917, **79,** 997.

153. Davison, W. C., and A. S., and Miller, M. K.: Meningitis in an Infant Due to Hitherto Undescribed Organism, Micrococcus Florens. Jour. Exper. Med., 1917, **26,** 779.

Antimeningococcus serum was administered. Symptoms of meningitis appeared. The injections were continued. The fluid became almost clear but contained many cells.

On the twenty-ninth day of the disease there was an abrupt rise of temperature, and a reappearance of signs of acute meningitis. The eighteenth lumbar puncture yielded very cloudy fluid in which, for the first time, organisms were demonstrable in great abundance in both films and cultures. They were gram-negative intracellular and extracellular diplococci, many of them arranged in groups of twos and threes. They continued to be present in the greatest abundance, and, later, were isolated also from the blood stream. The child's condition became worse and he died on the forty-fifth day of the disease.

The bacteriology of the gram-negative micrococcus repeatedly isolated from the spinal fluid of this infant did not correspond in its reactions to that of any known organism. Details are given. Because of its luxuriant growth in all mediums tested the organism has been tentatively called *Micrococcus florens*. Tests of its virulence lead to the belief that it is pathogenic for mice, rabbits and monkeys, producing a septicemia and also, in the two latter animals, causing a purulent meningitis. The combined results of the biologic reactions and of the agglutination tests lead to the belief that the organism, though morphologically similar to the meningococcus, is yet entirely distinct.

G. Rutelli[154] describes the case of a girl, aged 6 months, admitted with symptoms of cerebrospinal meningitis. Ten c.c. of fluid obtained by lumbar puncture was turbid and formed dense white clots after a few minutes. The microscope showed numerous white cells, the neutrophil polynuclear leukocytes being prevalent, several erythroblasts and abundant filamentous organisms resembling streptothrix. An emulsion of this organism injected into the peritoneum of some white mice caused death in three days. The patient's blood did not agglutinate them. A second lumbar puncture gave identical results. Hexamethylenamin was given in doses of 1 gm. daily. The temperature continued irregular and intermittent but never rose above 38.5 C. The sequal of the case is unknown, as the parents removed the patient from the hospital.

Meyer[155] reports the case of a girl, aged 6 years, an Indian child, and her brother, aged 8 years, and sister, aged 12 years. All showed varying degrees of distinct meningeal symptoms. The first mentioned had been ill six days with headache and vomiting. Rigidity of the

154. Rutelli, G.: Cerebrospinal Meningitis from Streptothrix. Pediatria. 1915, **23,** 711.

155. Meyer, J.: Trichinosis: A Report of Three Cases Simulating Meningitis, with the Findings of Trichinae Larvae in the Spinal Fluid. Jour. Am. Med. Assn., 1918, **70,** 588.

neck and photophobia were present. Kernig's sign was positive, the knee jerks were absent. In two of the cases trichinae larvae were found in the spinal fluid. There was marked cellular increase (lymphatic) in the spinal fluid. There was general leukocytosis and eosinophilia, 8 per cent. A clinical resemblance to poliomyelitis was noted. The finding of trichinae in the spinal fluid was first reported by Van Cett and Lintz (1914) and since then their observations have been confirmed. The presence of meningeal symptoms has not been emphasized.

Baeslack, Bunce and co-workers[156] report the cultivation of the meningococcus from the blood. That the meningococcus occasionally gets into the blood is borne out by the occurrence of lesions in various parts of the body from which meningococci are recovered. Such a case was noted in this review last year.[157] A somewhat analagous finding is noted later (Netter, Selanier, etc.). By placing 15 or 20 c.c. of the patient's blood in each of two bottles containing from 80 to 100 c.c. of meat infusion bouillon, salt-free, to which had been added, after sterilization, 1 per cent. glucose, and 10 per cent. serum water, then incubating at 37 C., Baeslack, Bunce and co-workers have been able to obtain 36.3 per cent. positive cultures.

As the result of their study of twenty-five cases they conclude that systemic infection by the meningococcus is more frequent than previously suspected; that this systemic infection may occur without appreciable or with no meningeal localization; and that the systemic infection may be previous to, or coexistent with, meningeal involvement. Consequently, the intravenous administration of antimeningococcus serum is rational and is indicated in conjunction with the intraspinal treatment.

A second communication by Netter, Salanier, and Wolfrom,[158] a paper read before the Société de Biologie, Paris, Nov. 18, 1916, reviews the work of the last two years bearing on the recognition of meningococci in the purpuric eruptions that may accompany obvious cases of meningococcal meningitis, or precede the meningeal involvement, or even exist apart from any symptoms of meningitis whatever. These authors claim precedence. in that whereas other examinations had been made postmortem, theirs were made *during the patient's life*—a matter

156. Baeslack, F. W., Bunce, A. H., Brunelle, G. C., Fleming, J. S., Klugh, G. F., McLean, E. H., and Salmon, A. V.: Cultivation of Meningitis Intracellularis from the Blood; Preliminary Report. Jour. Am. Med. Assn., 1918, **70,** 684.

157. Holmes, James B.: Recent Work in Anatomy, Physiology and Pathology of Childhood. Am. Jour. Dis. Child., 1917, **13,** 198.

158. Netter, A., Salanier, M., and Wolfrom: A Case of Hyperacute Purpura without Cerebrospinal Meningitis; Recognition of Its Meningococcal Nature During Life by Microscopic Examination. Brit. Jour. Child. Dis., 1917, **14,** 104.

of practical importance from a diagnostic point of view. Benda, Pick, Sharpe, Ghon and Barber are the names mentioned. They report a fresh case of primary meningococcal purpura whose nature was revealed during life by microscopic examination of the skin lesions. A 10-month-old infant had been seized with shivering and fever, followed by convulsive movements one afternoon. It was still feverish throughout the night, and next morning its mother found it had large violet spots all over the body. The child was extremely weak, dyspneic, and cyanosed, but free from signs of meningeal reaction. Lumbar puncture yielded normal spinal fluid. Scarification of the darkest purpuric patches yielded sanguinolent fluid, and in the smears of this serum were found definite and characteristic meningococci. The child died.

Miscellaneous.—Blackfan[159] reports 4 cases of lead poisoning in children. Convulsions were a prominent symptom. The knee kicks were absent; Kernig's sign was not present; the reflexes were not exaggerated. In three of the four cases, changes were found in the spinal fluid—a positive globulin reaction, or an increase in the number of cells, or both. Three of the patients died, two in the hospital and one in a succeeding attack three weeks after an uneventful recovery and discharge. The source of the lead was usually in paint gnawed from the crib or other furniture.

Stewart,[160] in 1895, reported a series of convulsions due to eating buns colored with chrome yellow. Seven of the 16 cases were in children between 1½ and 12 years of age. Numerous subsequent reports (Gibson, Love, Turner, Breinl and Young) have come from Australia where there has been an unusually good opportunity to study this condition. As Blackfan says:

> In all patients with convulsions in which the etiological factor is not clear, lead should be suspected. This can readily be determined, as in the majority of instances there are other evidences of the condition, e. g., the lead line, basophilic degeneration, and the presence of lead in the feces. The examination of the spinal fluid may prove to be an index as to the seriousness of the affection and of prognostic aid.

Several papers dealing with diseases of the muscles may be noticed: McCrudden[161] says that the myasthenia of progressive muscular dystrophy is due to hypoglycemia, and the hypoglycemia and fat deposition to impair glycogenesis, the carbohydrate of the food being probably changed largely to fat instead of glycogen. The impaired glycogenesis is due to suprarenal or other endocrine disease.

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159. Blackfan, K. D.: Lead Poisoning in Children, with Especial Reference to Lead as a Cause for Convulsions. Am. Jour. Med. Sc., 1917, **153,** 877.

160. Stewart: Lead Convulsions. Am. Jour. Med. Sc., 1895, **59,** 288.

161. McCrudden, F. H.: Progressive Muscular Dystrophy. Jour. Am. Med. Assn., 1918, **70,** 1216.

This view is based on the following data: In progressive muscular dystrophy there is found a low blood sugar—and following treatment which increases the blood sugar, an improvement in muscular strength; blood sugar—glucose—is the source of energy for muscular contraction, and agencies which reduce blood sugar. Addison's disease, diphtheria toxin, and phosphorus and hydrazin poisoning, cause profound myasthenia, the severity of which is proportional to the fall in blood sugar; there is no increased utilization of sugar, shown by the absence of rise in heat formation, and no loss by the kidneys, the urine being free from sugar; there is in progressive muscular dystrophy a rapid fall in blood sugar during the first twenty-four hours of starvation—in health the normal blood sugar level is maintained during starvation—which testifies that in progressive muscular dystrophy as in all forms of experimental hypoglycemia there is a decreased rate of replenishment resulting from a diminished reserve of glycogen; the diminished glycogen reserve, in experimental hypoglycemia, results from impaired glycogen formation, the carbohydrate not being converted into glycogen, but remaining a long time in the blood, after which, judging from the amount of "fatty degeneration" in these cases, from the increased respiratory quotient, and the lipemia, it is probably changed to fat—the deposition of fat in the muscles may be so great as to lead to the use of the term hypertrophic; impaired glycogenesis may result from damage either to the liver, the organ in which glycogenesis takes place, or to the suprarvenals (or other endocrine glands), the organs that control the process; a prompt and marked rise in blood sugar following the administration of epinephrin in progressive muscular dystrophy testifies to the efficiency of the liver in the disease and points to a deficiency in epinephrin.

Reuben[162] reports six cases of amyotonia congenita (Oppenheim), and gives a full review of the literature.

Cano[163] writes of reflex amyotrophy.

Crafts[164] reports three cases of myasthenia gravis.

Navarro and Correas[165] consider the pathologic histology of the muscles in cases of severe myopathy.

Johannessen[166] writes on progressive multiple ossifying myositis.

Tuberculosis.—Clough[167] reports the cultivation of tubercle bacilli from the circulating blood of a 5-year-old colored boy suffering from

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162. Reuben, M. S.: Arch. Int. Med., 1917, **20,** 657.
163. Cano, J. O., Rev. de med. y cirug., Havana, 1918, **23,** 59.
164. Crafts, L. M.: Journal-Lancet, 1917, **37,** 774.
165. Navarro, J. C., and Correas, C. A.: Semana méd., 1917, **24,** 628.
166. Johannessen, C.: Norsk Mag. f. Lægevidensk., 1917, **78,** 839.
167. Clough, Mildred C.: The Cultivation of Tubercle Bacilli from the Circulating Blood in Miliary Tuberculosis. Am. Rev. Tuberc., 1917, **1,** 598.

miliary tuberculosis. The occurrence of tubercle bacilli in the circulating blood in miliary tuberculosis has long been recognized. The pathologic anatomy of the disease indicates that tubercle bacilli must be distributed throughout the body from a primary focus by means of the blood stream. Faber (1914) obtained a growth of tubercle bacilli on a plain agar slant inoculated with blood from the heart of a child dead of generalized tuberculosis. Clough for the first time has grown tubercle bacilli from the circulating blood of living patients. By her method positive cultures have been obtained from the blood in a relatively short time. The possibility of using the procedure in differential diagnosis is thus suggested.

Wang and Cooley[168] have taken up the occurrence of tubercle bacilli in the breast milk of tuberculous women. Behring, in 1903, announced that pulmonary tuberculosis could result from intestinal infection and that infants often became tuberculous from the milk fed to them. Marfan mentioned two cases which he considered authentic examples of children being infected by their mother's milk and Kurashiga found tubercle bacilli in the breast milk of tuberculous women who had no mammary disease. Noeggerath was able to infect guinea-pigs with tuberculosis by injecting them intraperitoneally and subcutaneously with breast milk. He did not believe tubercle bacilli to be excreted in breast milk in large enough numbers to be a frequent and large source of danger to the child, but thought it best, nevertheless, for tuberculous mothers to feed their children artificially.

Wang and Coonley examined the breast milk of twenty-eight women. They made smears for tubercle bacilli which were negative. They injected guinea-pigs intraperitoneally with negative results. They also fed guinea-pigs the milk, thinking to see if it contained any toxic substance that might prevent thriving. Both pigs lost weight but showed no evidence of tuberculosis. With one exception, which may be questionable, the results of their studies were negative.

During recent years much work has been published tending to show the relatively great amount of infection with bovine tubercle bacilli in Edinburgh as compared with similar infection in American cities. The work of Park and Krumwiede has shown that in New York City, at least, tuberculosis in children is usually, indeed almost always, of human origin; in Edinburgh, on the contrary, a very large percentage is of bovine origin. This difference in type of infection has been attributed to the use, respectively, of pasteurized milk from tuberculin tested cows on this side and raw, less well tested milk in Scotland.

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168. Wang, S. L., and Coonley, F.: Occurrence of Tubercle Bacilli in Breast Milk of Tuberculous Women. Jour. Am. Med. Assn., 1917, **69**, 531.

Mitchell[169] has recently published some interesting observations from Edinburgh on primary tuberculosis of the faucial tonsils obtained from children with tuberculosis of the upper cervical lymph nodes, and from children with enlarged tonsils only and without evidence of tuberculosis in the cervical glands or elsewhere.

In the first group were 100 children and 6 adults. In no case was it possible to establish the presence of tuberculosis prior to removal and examination of the tonsils (except doubtless through the von Pirquet test). The tonsils were rarely painful, and there were no symptoms except swelling of the upper cervical nodes, which in about one-fourth of the cases was slight, and in the rest rather extensive. On microscopic examination of these 106 tonsils, 41, or 38 per cent. of the tonsils were definitely tuberculous. Animal inoculations in 92 cases, yielded 20 positive results, the bovine type of bacillus being isolated in 16.

In the second group, there were 100 cases, all children with enlarged tonsils only. The cervical glands were palpable in all. Microscopically, 9 of the tonsils were tuberculous also, all 9 yielding positive results on inoculation. The type of organism was in 4 bovine, in 2 human, and in 3 undertermined.

The tubercles in the tonsils were situated near the deeper portions of the crypts, and also directly beneath the surface mucosa as well as deep in the tonsil close to the capsule, the first two being the favorite locations. The fact that the majority of the tuberculous foci were in positions within easy reach of the oral cavity suggests infection by way of the mouth. The absence of demonstrable pulmonary lesions in these cases would seem to preclude infection of the tonsils from the sputum. The high proportion of organisms of the bovine type recovered—80 per cent. in the positive inoculations in the first series and 66 per cent. of those recognized in the second series—would point strongly to the milk supply as the source of infection in primary tuberculosis of the tonsils. This work should stimulate further studies. It is to be noted that the children came from the dispensary class.

Wang[170] has studied the types of the bacilli in children at necropsy in Edinburgh. He gives the usual high bovine origin for tuberculosis in that region; 50 per cent. in children, 13 per cent. in adults; and hence concludes that it is often of intestinal origin.

Spleen and Blood.—De Lange and Schippers[171] report two cases

169. Mitchell, A. P.: Primary Tuberculosis of the Faucial Tonsils, Jour. Path. and Bact., 1917, **21,** 248.

170. Wang: Jour. Path. and Bact., 1917, **21,** 131.

171. De Lange, C., and Shippers, J. C.: Familial Splenomegaly. Am. Jour. Dis. Child., 1918, **15,** 249.

which presented the clinical picture of splenomegaly of the Gaucher type. A study of the blood pictures presented by the brothers and sisters of the patients and their parents and grandparents left these workers with "the distinct impression that in such cases an examination of the family ought to be carried out as carefully as possible," though they would not actually state that in this instance the origin of the disease in the children could be traced back to the father. Splenectomy was successfully performed in both children. In the first child, 5½ years old, the spleen weighed 170 gm. (about five times the normal weight); in the second child, 7½ years old, the spleen weighed 470 gm. (eight times the normal weight). The splenic and lymphnodular follicles showed very great activity of the germinal centers, and karyokinetic figures in the pulp. In both spleens a number of sudanophil cells were inside and outside of the follicle. These, however, are not to be looked on as abnormal, as Kusunoki[172] found these cells regularly in his examination of 132 spleens, and according to him lipoid cells are found in the follicles of the spleen in children, whereas in adults they are seen in the pulp.

Though the cases presented the clinical picture of Gaucher's disease, the microscopic pictures bore no resemblance to the pathologic findings of that disease—an opinion confirmed by Dr. R. de Josselin de Jong. Apparently one had to deal with a congenital *vitium primae formationis;* on account of which the spleen developed into a state of "giant growth." And certainly one had to deal with "a familial progressive disease of the spleen which produces, sooner or later, an increasing cachexia, and in the course of which a hemorrhagic diathesis occurs antemortem."

The present status of idiopathic splenomegaly (Gaucher's Disease) is considered in a paper by Vaglio.[173]

Holsclaw[174] reports a case of acute lymphatic leukemia in a 11-month-old Japanese baby. The family history and the child's previous history were negative. She was sent into the hospital with a diagnosis of adenitis; a swelling of the glands of the neck had been noticed ten days earlier.

It was found that the mucous membranes were extremely pale, and the gums hemorrhagic. The glands were everywhere palpable. They formed a complete collar about her neck, and were hard, nonfluctuating, and discrete, not tender. The spleen was not markedly enlarged. Hemoglobin, 30 per cent.; red cells, 3,200,000; white cells, 109,000;

172. Kusunoki: Zieglers Beitr. z. path. Anat. u. z. allg. Path., 1914, **59.**

173. Vaglio, R.: Present Status of Idiopathic Splenomegaly (Gaucher's Disease). Pediatria, Naples, 1917, **25,** 639.

174. Holsclaw, F. M.: A Case of Acute Lymphatic Leukemia, with Autopsy Arch. Pediat., 1918, **35,** 151.

chiefly small lymphocytes. There was irregular fever. The child died on the nineteenth day of observation, following a transfusion. The microscopic findings were of acute lymphatic leukemia with slight infiltration of the liver and kidneys.

A case of pseudoleukemia infantum (von Jaksch's disease) with essentially negative necropsy findings is reported by Roth.[175]

Bass[176] reports a case of Winckel's disease. The combination of cyanosis, jaundice, and hemoglobinuria in the new-born is a very rare one, only about thirty cases having been recorded up to 1911 (Brandenburg), though the syndrome was first described by Parrot in 1873, and by Winckel in 1879. It is generally thought to be infectious in origin, and various micro-organisms (*B. coli,* streptococcus, pseudo-diphtheria bacilli) have been claimed as the cause of the illness. The umbilicus is apparently not the portal of entry. The disease may occur epidemically in obstetric wards. Necropsy could not be obtained in Bass' case.

Lypodystrophia.—F. Parkes Weber presents two papers on Lypodystrophia Progressiva. In one[177] he reviews the published cases of this disease in females and tabulates them. He concludes that "lipodystrophia progressiva" is not always *progressive,* certainly not in regard to the extent of the area affected; that it usually commences in childhood, especially at from 6 to 8 years of age, that the disease or syndrome is not confined, as at first it was supposed, to the female sex; that the etiology is unknown, but is probably connected with a disorder of the internal secretions (endocrinic origin); and finally, that the fat atrophy, however disfiguring it may be, does not signify any danger to life, and is not usually accompanied by loss of strength and general health.

In the second paper[178] Weber reports the case of a 13-year-old English boy who was moderately fat in the face and body till the age of 8, after which loss of subcutaneous fat gradually progressed in the face and neck, and to a large extent in the arms, thorax, and abdomen. In the face, practically no fat was left save in the orbits. Ten other cases in males are tabulated; in seven, the affection appeared at from 2½ to 10 years of age. It appears that in males the fat atrophy is more often limited to the face and neck than in females.

The Severn Apartments.

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177. Weber, F. Parkes: Lypodystrophia Progressiva. Brit. Jour. Child. Dis., 1917, **14,** 81.

178. Weber, F. Parkes: Lypodystrophia Progressiva in a Male. Brit. Jour. Child. Dis., 1917, **14,** 179.

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